Supreme Court, U.S.
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No.

In The
SUPREME COURT OF THE UNITED STATES

October Term 1998

Randon Bragdon, D.M.D.,
Petitioner,

v.

Sidney Abbott, et al.,
Respondents.

ON PETITION FOR A WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE FIRST CIRCUIT

PETITION FOR WRIT OF CERTIORARI

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QUESTIONS PRESENTED

1. Under the Americans with Disabilities Act ("ADA"), are health care workers required by law to confront greater risks than patients?

2. Under the ADA, are a defendant's private expert's calculations of the statistical likelihood of a fatal injury, relying on data available in September 1994, admissible to raise a genuine issue of fact on whether an objectively reasonable dentist could have deemed a risk significant in September 1994?

3. Are factual and opinion assertions made for the first time on appeal in an amicus brief by a professional organization admissible evidence justifying summary judgment against a defendant on the issue of direct threat under the ADA?

4. Was it plain and highly prejudicial legal error for the Court of Appeals to maintain that 1987 CDC guidelines inform dentists universal precautions eliminate the need for additional precautions during invasive procedures?

5. Did the Court of Appeals' method in deciding Dr. Bragdon raises no genuine issue for trial so depart from accepted and usual standards of judicial review, and the prior decision of this Court, that its decision on remand should be vacated and the case remanded for trial?

6. Under Title III of the ADA, who has the burden of proof on the issue of direct threat?

7. What level of risk is legally significant under the direct threat provisions of the ADA?

LIST OF PARTIES

The parties below were plaintiffs/appellees Sidney Abbott and the Maine Human Rights Commission, and defendant/appellant Randon Bragdon, D.M.D.

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JURISDICTION

The judgment of the United States Court of Appeals for the First Circuit (the "Appeals Court") was entered on December 29, 1998 (10a). This Court has jurisdiction pursuant to 28 U.S.C. § 1254(1).

RELEVANT STATUTES AND REGULATIONS

42 U.S.C. § 12182(b)(3): "Specific Construction. Nothing in this subchapter [title III of the ADA] shall require an entity to permit an individual to participate in or benefit from the goods, services, facilities, privileges, advantages and accommodations of such entity where such individual poses a direct threat to the health or safety of others. The term 'direct threat' means a significant risk to the health or safety of others that cannot be eliminated by a modification of policies, practices, or procedures or by the provision of auxiliary aids or services."

28 C.F.R. § 36.208(c): "In determining whether an individual poses a direct threat to the health or safety of others, a public accommodation must make an individualized assessment, based on reasonable judgment that relies on current medical knowledge or on the best available objective evidence, to ascertain: the nature, duration, and severity of the risk; the probability that the potential injury will actually occur; and whether reasonable modifications of policies, practices, or procedures will mitigate the risk."

STATEMENT OF THE CASE

On September 16, 1994, Sidney Abbott visited Randon Bragdon, D.M.D., at the urging of the Executive Director of the Eastern Maine AIDS Network (485a, 487a). Ms. Abbott informed Dr. Bragdon she had been HIV positive for approximately eight years (259a). Dr. Bragdon performed a non-invasive dental examination on her in his office. He found a cavity near the gum line on a back lower tooth that needed filling (256a-259a). Filling the cavity required invasive procedures (577a-578a), which were defined in 1987 by Centers for Disease Control and Prevention ("CDC") as the manipulation or cutting of "oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists" (459a). According to the 1987 CDC guidelines, universal blood and body-fluid precautions "should be the minimum precautions for **all** such invasive procedures" (emphasis in original) (459a).

Based on this information and other evidence available in September 1994 (e.g., studies of the efficacy of universal precautions (226a, 400a, 414a, 418a, 466a, 623a, 637a), reported rates of accidental sharps injuries among dentists (316a, 372a), published seroconversion rates after percutaneous HIV exposure (318a, 557a), OSHA analyses (e.g., 56 Fed. Reg. at 64092), and other CDC guidelines and reports of actual and possible HIV transmissions (224a, 549a-550a), Dr. Bragdon believed performing the invasive procedures necessary to fill Ms. Abbott's cavity posed a direct threat of accidental HIV transmission to himself and his staff, meaning, a significant risk that could not be eliminated by universal precautions, by other reasonable modifications of policies, practices, or procedures, or by the provision of auxiliary aids or services. *See* 42 U.S.C. § 12182(b)(3) (definition of direct threat).

Dr. Bragdon believed the best way to minimize the HIV risk consistent with the highest standard of care for Ms. Abbott, was to perform the invasive procedure using

state-of-the-art infection control in the most aseptic environment available. He therefore offered to fill the cavity in what he thought would be a local hospital, less than 1.5 miles from where Ms. Abbott lived and worked, and where he believed dental facilities were maintained and he would be permitted to perform the procedure "without any problem" (261a, 265a, 584a). Ms. Abbott said she would consider it (579a), but instead she sued him under Title III of the ADA. The District Court had jurisdiction under 28 U.S.C. § 1331.

The District Court granted summary judgment against Dr. Bragdon based on affidavits of a public health official that were not available until after September 1994. *Abbott*, 912 F. Supp. at 589. The Appeals Court, and later this Court, agreed the affidavits could not be used against Dr. Bragdon in determining whether Dr. Bragdon's risk assessment was objectively reasonable from the standpoint of medical and objective evidence available in September 1994 (22a). The Appeals Court nonetheless affirmed summary judgment against Dr. Bragdon based on its reading of 1993 CDC Dentistry Guidelines (the "1993 Guidelines") and a 1991 American Dental Association Policy Statement on HIV (the "1991 Dental Association Policy") (44a-45a). The Court granted certiorari.

By opinions issued June 25, 1998, four members of the Court agreed "it is clear . . . that [Dr. Bragdon] presented more than enough evidence to avoid summary judgment on the 'direct threat' question" (Part II of Rehnquist, C.J., concurring and dissenting in part, joined by O'Connor, J., Scalia, J. and Thomas, J.) (30a, 32a).

Three members of the Court were uncertain (Kennedy, J., Souter, J., and Ginsburg, J.). They believed CDC, or other public health authority guidance available in September 1994, should be given special weight in understanding what would be an objectively reasonable risk assessment at the time, and that the existence of a significant risk must be determined from the standpoint of

objectively reasonable dentists. However, because the 1993 Guidelines "do not assess the level of risk," they doubted the 1993 Guidelines justified summary judgment against Dr. Bragdon (21a-23a).

The same members of the Court questioned the Appeals Court's reliance on the 1991 Dental Association Policy. They pointed out the American Dental Association is not a public health authority, and that the ADA calls for a "scientific assessment of the risk," which is a question of "statistical likelihood, not professional responsibility." They thought the record did not disclose a scientific or statistical basis for the 1991 Dental Association Policy. The Policy was some evidence the medical community had assessed the risk, but it did not prove there was no direct threat, or that no reasonable dentist could have thought there was a direct threat based on other medical or objective evidence (23a-24a). Furthermore, although Dr. Bragdon and Ms. Abbott presented affidavit testimony from expert witnesses, the affidavits did not disclose whether the opinions were based on information available in September 1994 (24a). They therefore favored vacating the judgment and remanding to "permit a full exploration of the issue" (26a). Justice Ginsburg emphasized it was best to err, if at all, "on the side of caution," and her stated goal was to "ensure a fully informed determination" (28a).

Justices Stevens and Breyer thought summary judgment should be affirmed. However, to make a majority of five, they joined Justices Kennedy, Souter, and Ginsburg in favoring a remand to the Appeals Court (27a). The effect was to block a remand for trial, even though of the six justices who expressed an opinion on the issue, four thought Dr. Bragdon clearly raised genuine issues for trial.

The Appeals Court's decision on remand, *see* 5a-6a, relies on CDC's 1987 *Recommendations for Prevention of HIV Transmission in Health-Care Settings*, 36 MMWR No. 2S (Aug. 21, 1987) (the "1987 Guidelines") (453a-461a.13), instead of CDC's 1993 Guidelines (527a-547a). Such

reliance is so literally misplaced it is hard to imagine what the Appeals Court was thinking. The decision also applies a legally unjustified double standard, inexplicably ignores Dr. Bragdon's expert's statistical calculations, and wrongly treats as undisputed certain factual and opinion assertions made by the American Dental Association for the first time on remand in an amicus brief.

The Appeals Court's opinion is so deeply flawed that it cannot be said to have accomplished the tasks set by the Court of a full exploration of the scientific evidence and a fully informed determination. The opinion, at best, carries forth *sub silentio* a policy determination that regardless of the scientific and statistical evidence available to dentists in September 1994, and regardless of what CDC publications actually say, the case should not go to trial.

REASONS FOR GRANTING THE WRIT

I. THE COURT SHOULD EXERCISE ITS SUPERVISORY POWER BECAUSE THE APPEALS COURT'S DECISION IS SO PLAINLY FLAWED THAT IT DOES NOT ACCOMPLISH THE GOAL SET BY THE COURT OF A FULLY INFORMED DETERMINATION OF THE ISSUE.

A. The 1987 CDC Guidelines Literally Say The Opposite Of What The Appeals Court Says They Say.

The Court properly doubted the 1993 Guidelines support summary judgment on the level of risk (23a). Thus, on remand, the Appeals Court relied on the 1987 Guidelines, instead of the 1993 Guidelines. The Appeals Court says the 1987 Guidelines "explain[] that the use of the universal precautions eliminates the need for additional precautions that CDC formerly had advocated" (6a). This information from the 1987 Guidelines, according to the Appeals Court, fills the implicit gap in the 1993 Guidelines, thereby informing objectively reasonable dentists there is no significant risk, thereby proving Dr. Bragdon acted irrationally.

Under a bold heading "**Precautions for Invasive Procedures,**" the 1987 Guidelines define an "invasive procedure" as, *inter alia*, "the manipulation . . . of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists" (459a). Filling a cavity near the gum line is a quintessential example of an invasive procedure (577a-578a). The 1987 Guidelines then say:

> The universal blood and body-fluid precautions listed above, combined with the precautions listed below, should be the minimum precautions for **all** such invasive procedures.

(459a) The "universal blood and body-fluid precautions listed above," to which the 1987 Guidelines refer, are routine precautions for non-invasive procedures. They take the place of formerly recommended CDC isolation precautions in non-invasive settings (459a). The "precautions listed below," to which the 1987 Guidelines refer, are special precautions for invasive procedures, for dentistry, and for other specialized services (459a). The 1987 Guidelines say the precautions listed above and below **combined** should be the **minimum precautions for all invasive procedures**. The Appeals Court says the 1987 Guidelines say the opposite, namely, universal precautions "eliminate the need for additional precautions." On that basis, and really only on that basis, the Appeals Court rules that Dr. Bragdon is a civil rights violator who does not deserve a trial, and who should be exposed for liability under the ADA for Ms. Abbott's attorneys' and experts' fees in excess of $300,000.

What is going on?

The reasonable thing to conclude from the 1987 Guidelines, since it is what they say, is that universal precautions, plus all the other precautions described in the Guidelines, are ***not*** the maximum prudent precautions when filling cavities near the gum line, but **the minimum**

precautions. This clearly expresses the view that even with universal precautions, there is a residual risk of significance, and therefore it is wise to take additional precautions, and in fact dentists are told they "should" take additional precautions. Yet the Appeals Court says the opposite, and based on that, affirms summary judgment against Dr. Bragdon again.

With this kind of topsy-turvy, up-is-down logic, what could Dr. Bragdon possibly have said to convince the Appeals Court that published CDC guidelines do not support Ms. Abbott and there is a genuine issue for trial? What could he have said? What's more, Dr. Bragdon specifically pointed out in his response brief that the 1987 Guidelines support him, not Ms. Abbott, on this very point (670a-673a). The Appeals Court accepted the brief for filing (684a). In addition, how are conscientious professionals in the future supposed to understand what the law requires, when the law as it now stands is that CDC guidelines mean the opposite of what they actually say? It just does not get any more Orwellian than this.

There is no way the highest court in any country should let this stand as a fully informed determination of an issue. To do so would insult the integrity of the judicial process and trivialize in the extreme the Court's previous decision to vacate and remand.

B. The Appeals Court Inexplicably Ignores Dr. Bragdon's Expert's Calculation Of The Risk Based On Objective Information Available In September 1994.

Throughout the case, and on remand, Dr. Bragdon argued that his expert witness, Sanford Kuvin, M.S., M.D., D.T.M.&H., calculated the risk, by accepted scientific methodology, of contracting HIV by accidental needlestick when giving intra-oral injections to HIV-positive patients. The risk is 1 in 67,000 per intra-oral injection, or 1 in 10, over 30 years of practice, if a dentist gives one such injection each working day (606a). A 1 in 67,000 risk is

greater than the risk of a fatal accident while skydiving, which is approximately 1 in 100,000 (592a, 596a).[1]

On remand, Dr. Bragdon further emphasized how, according to the Court, the "question under the [ADA] is one of statistical likelihood, not professional responsibility" (24a, 83a-86a). Dr. Bragdon explained in careful detail in his brief, *see* 91a-119a, 147a-151a, why Dr. Kuvin's calculations of the risk are reliable and admissible, how they are based on published data available in 1994, why Dr. Kuvin's results comport with CDC estimates of the risk to patients (106a-112a), why his results comport with the number of documented and possible occupational transmissions of HIV as reported by CDC's "passive surveillance" system (147a-151a), and why his results raise a genuine issue for trial (116a-119a).

The Appeals Court ignored Dr. Kuvin's analyses with no explanation at all. There is no hint as to whether the Appeals Court thought Dr. Kuvin's analyses were inadmissible, or if admissible, not on point, or if on point, insufficient to raise a genuine issue of the statistical likelihood of the risk, or if sufficient to raise that issue, insufficient to raise a genuine issue that that level of risk is legally significant.

In sharp contrast, the Appeals Court states Ms. Abbott "proffered the opinions of several prominent experts" (7a). These are the same, non-public health authority, private expert opinions referred to by this Court, when it noted that it could not tell if such opinions were based on information available in September 1994 (24a). To correct this deficiency, Dr. Bragdon moved on remand for a further, limited remand, to give experts a chance to clarify through affidavits the extent to which their opinions are based on

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[1] OSHA assesses the significance of a risk based on an assumed 45 years of exposure during an entire working lifetime. *See* 56 Fed. Reg. at 64028. Dr. Kuvin's calculation based on 30 years of exposure is therefore conservative by accepted OSHA standards.

information that was available in September 1994 (55a). Dr. Bragdon was particularly concerned because previously the Appeals Court in a footnote ostensibly discounted Dr. Kuvin's analyses because the Appeals Court was uncertain that Dr. Kuvin relied on information available in September 1994 (45a-46a). Dr. Bragdon wanted to make sure the record is clear that Dr. Kuvin's calculations and opinions rely on data that was available to knowledgeable dentists in September 1994.

The Appeals Court denied Dr. Bragdon's motion which would have given the experts a chance to clarify whether their opinions were based on information available in September 1994 (684a). The Appeals Court then applied a double standard whereby it ignored the expert opinion of Dr. Kuvin (e.g, 573a-574a, 606a), but cited Ms. Abbott's experts' opinions as further evidence against Dr. Bragdon, with nothing more to rely on than what the Court rejected as too uncertain to rely on (7a).

The rule at summary judgment is that courts must view the record in the light most favorable to the non-movant. What the Appeals Court did by nullifying Dr. Kuvin without comment, while citing Ms. Abbott's private experts, is totally contrary to the rule of law. This further illustrates how unspoken policy judgments are controlling this case, rather than the law or any objective evidence available to dentists in September 1994.

C. The Appeals Court Ignored Evidence That Universal Precautions Have Never Been Shown To Be Effective Against HIV.

Dr. Bragdon explained in detail, *see* 120a-127a, why no knowledgeable dentist in 1994 could have concluded universal precautions have any significant impact on the risk of contracting HIV in the dental office. *See also, e.g.*, 226a; Reingold, et al., *Failure of Gloves and Other Protective Devices to Prevent Transmission of Hepatitis B Virus to Oral Surgeons*, JAMA, vol. 259, no. 17 (1988) (400a); Noble, et al., *Hepatitis B and HIV infections in*

dental professionals: Effectiveness of infection control procedures, J. Can. Dent. Assoc., vol. 57, no. 1 (1991) (418a). A corollary is that no knowledgeable dentist in 1994 would have concluded universal precautions are the best way to combat the risk of HIV transmission when working on a person known to carry HIV (*see* 127a-130a). This evidence was also ignored by the Appeals Court.

The real effect of universal barrier precautions is to provide some extra safety at the statistical margins against the transmission of many diseases including HIV, while at the same time placating irrational public fears about HIV. In truth, the statistical risk of HIV transmission during invasive medical procedures is *less* than many irrational people probably think, but much *more* than CDC leads people to think, probably on account of policy-guided fears that admission by authorities of any non-trivial risk will fuel irrational patient behavior, which will lead to more people dying due to not going to doctors for fear of HIV, than would die by accidental HIV transmission. For policy purposes, public health officials rationalize the view that the HIV risk to dentists is acceptably low, since, in their minds, no practical alternative eliminates the known risk of percutaneous injuries, short of not performing the procedures at all, which is unacceptable.

Likewise, CDC scientists know there is no scientific evidence that universal precautions significantly reduce the risk of HIV transmission. So do American Dental Association scientists (414a-417a). That is why the Dental Association *opposed* OSHA's 1991 bloodborne pathogen regulations, which require dental employers at costs in excess of $20,000 a year to maintain universal precautions for employees (431a). *See American Dental Ass'n v. Martin*, 984 F.2d 823 (7th Cir.), *cert. denied*, 510 U.S. 859 (1993)). Only if "universal precautions" are defined to include the hepatitis B vaccine (which they do under OSHA regulations and which legally justified the regulations), have "universal precautions" been shown to reduce any

significant risks. But that is the effect of the vaccine, not gloves, masks, and plastic wrap.

D. The Appeals Court Misstates The Record When It Says The Origins Of The American Dental Association's 1991 Policy On HIV Are Undisputed, And The Appeals Court Improperly Admitted As Evidence Against Dr. Bragdon Claims Made By The Dental Association For The First Time In Its Amicus Brief On Remand.

In its first opinion, the Appeals Court seemed to accord public health authority status to the American Dental Association's 1991 Policy on HIV (the "1991 Policy" or "Policy"). This Court held, however, that the American Dental Association is *not* a public health authority and the scientific basis of the 1991 Policy is unclear (24a). For example, the Policy refers to "current scientific and epidemiologic evidence," but does not identify the evidence. The mere existence of the Policy, therefore, did not justify summary judgment against Dr. Bragdon (24a).

On remand, the Appeals Court asked the parties to brief, *inter alia*, "the import and admissibility of the . . . 1991 American Dental Association Policy on HIV" (53a-54a). Dr. Bragdon then learned the Dental Association planned to file an amicus brief. It took no genius to predict the Dental Association would use its amicus brief to make self-serving claims as to the basis of its Policy. Dr. Bragdon therefore moved for a limited remand to permit him to explore through normal discovery the actual historical and epistemological origins of the Policy (55a). Dr. Bragdon also asked the Dental Association to share with him voluntarily information about the origins of the Policy (506a, 509a, 512a). The Dental Association refused (513a).

In Dr. Bragdon's brief, he argued the alleged scientific opinion expressed in the 1991 Policy was inadmissible hearsay, as were any factual assertions about the historical development of the Policy made by the Dental Association

in an amicus brief (131a-135a). In the alternative, Dr. Bragdon argued that if the Policy is admissible expert testimony, it is entitled to less weight than a normal, Rule 702 expert opinion, since the Dental Association is not a public health authority, the real authors of the opinion are unknown, no specific data or studies are cited, there is no mathematical or statistical analysis, it is not subject to cross-examination, and OSHA earlier specifically rejected the American Dental Association's assessment of the HIV risk to dentists as "confusing and not supported by the data." 56 Fed. Reg. 64004, 64092 (Dec. 6, 1991). In other words, legally, why should a court require dentists to give credence to the Dental Association's 1991 Policy when OSHA expressly rejected the Association's reasoning on the HIV risk to dentists as confusing and not supported by the data? (134a-135a)

After the Dental Association filed its brief, which was Dr. Bragdon's first chance to see it, Dr. Bragdon renewed his motion to conduct discovery against the Dental Association (675a). He also pointed out numerous analytic gaps in the Dental Association's amicus presentation concerning the alleged scientific foundation of the Policy, including the Policy's misplaced reliance on the 1987 Guidelines (679a).

The Court denied Dr. Bragdon's motion (684a).

Dr. Bragdon renewed the motion at oral argument.

Three weeks later the Appeals Court issued its opinion stating the process by which the 1991 Policy was assembled is a matter of "undisputed fact," and that the undisputed facts show that the Policy has a scientific foundation (6a-7a).

How can that be? How can that be?

Dr. Bragdon disputed the historical and epistemological bases of the 1991 Policy at every step: He argued the Dental Association's claims were inadmissible, and if admissible, entitled to less weight than his own expert's opinion. He pointed out historical gaps in the account of

the genesis of the Policy, and he pointed out logical holes in the Dental Association's so-called "science." He tried repeatedly to remand for discovery to expose the true origins of the Policy. Yet the Appeals Court said the scientific basis of the 1991 Policy was "undisputed."

But even if the Dental Association's factual claims about the process were undisputed, which they are not, the Appeals Court still confuses scientific method with committee process (6a-7a). What this Court thought important was not the *ipse dixit* of the Dental Association, but any statistical or scientific basis for the Policy. The Dental Association's factual claim that the Policy was formulated by a group of experts (6a-7a) is not scientific proof of its validity, but merely the claim that the Policy is based on science. The question is, what science?

The Dental Association in fact offered no new scientific evidence or calculation of the risk. It also omitted crucial details. For example, it said its 1991 Policy was largely based on the 1987 Guidelines, but it did not explain how the 1987 Guidelines say universal precautions should be the minimum precautions for all invasive procedures. The Dental Association also vaguely described the testing of dentists for HIV at annual dental conventions, but provided no analysis of whether a statistically significant inference may be drawn. Dr. Bragdon has now learned from persons familiar with the process that the testing was voluntary, and each year a significant group of dentists chose (i.e., self-selected) not to be tested.

Thus, whereas Dr. Bragdon and OSHA rely on, for example, the scientifically acceptable Klein study, *see* 56 Fed. Reg. at 64092, which suggests to unbiased readers a significant incidence of occupationally acquired HIV among dentists, the Dental Association relies on what OSHA calls confusing contentions not supported by the data, *id.* Nevertheless, the Appeals Court credits the factual and opinion statements of the Dental Association in an amicus brief, says they are undisputed when they are disputed,

and says they also justify summary judgment against Dr. Bragdon. This is not science or law, and it does not accomplish the goals set by this Court on remand.

E. The Court's Previous Analysis Of The Probative Value Of Seven Possible Occupational Transmissions Of HIV Is Based On A Case Of Mistaken Identity.

Dr. Bragdon explained to the Appeals Court that this Court, in its original decision, confused the seven reported possible transmissions of HIV to dental workers reported by CDC in June 1994[2] with seven other dental workers reported by Gooch, et al.[3] (*see* 144a-147a). Gooch reported in 1995 that a total of seven dental workers who were enrolled in a CDC needlestick study did not report back for HIV testing to see if they seroconverted after percutaneous HIV exposure at work (*see* 339a (Gooch explaining that six dental workers did not return for follow-up testing after establishing baseline seronegativity, and a seventh was never tested)). The Court's original decision confuses these seven dental workers with the seven dental workers reported in the June 1994 CDC surveillance report (*see* 25a-26a). The CDC surveillance report describes dental workers who already contracted HIV. The seven individuals described by Gooch failed to return for follow-up testing to see if they contracted HIV. By confusing the two groups, the Court ends up by doubting the probative value for Dr. Bragdon of the seven cases described by Gooch (*see* 26a). Instead, the correct question is how would reasonable dentists have understood the seven cases reported by CDC in its June 1994 surveillance report, reproduced at 224a.

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[2] CDC, *HIV/AIDS Surveillance Report*, vol. 6, no. 1, p. 15, tbl. 11 (Mid-year ed. June 1994), reproduced at 224a.

[3] Gooch et al., *Percutaneous Exposures to HIV-Infected Blood Among Dental Workers Enrolled in the CDC Needlestick Study*, 126 J. Am. Dental Assn. 1237, 1239 (1995), reproduced at 335a, 339a.

From the standpoint of dentists in September 1994, the seven dental-worker cases reported in the CDC table at 224a were described in the Journal of the American Dental Association in July 1994 to dentists as

> cases in which occupational transmission cannot be proven, but is strongly suspected. These cases involve health care workers who are . . . found to be HIV infected, and who have provided histories of occupational HIV-related exposures.

Susan Beekmann & David Henderson, *Managing Occupational Risks in the Dental Office*, 125 J. Am. Dental Assn. 847, 848 (July 1994).

Likewise, during recent hearings before the United States Civil Rights Commission, CDC's Deputy Director of the National Center for HIV, STD & TB Prevention, Dr. Ronald Valdiserri, explained that after "**extensive** investigation by CDC," these HIV-infected health care workers were found not to have another risk behavior, and that is why the cases are "**probable**," but not "definite" (emphasis added) (682a-683a). Published CDC reports in 1994 did not use the word "extensive" to describe the investigation conducted by CDC, but simply said the workers "have been investigated" (224a). However, ***exactly like OSHA and other experts in the field***, *see* 142a-144a, Dr. Bragdon reasonably treated the cases as probable (i.e., more likely than not) occupational HIV transmissions to dental workers, regardless of the label applied by CDC. CDC's own published, official position in 1988 was that "possible" transmissions of HIV to dental workers (and there were only two, not seven, at that time)

indicated a real threat to dentists, ***see* 466a**.[4]

F. The Appeals Court Ignored Evidence That Before 1994, Dentists Were Repeatedly Warned The Risk Of HIV Transmission To Them Was Greater Than The Risk To Their Patients.

This Court held that the existence of a direct threat must be determined from the standpoint of the practicing professional (21a). Dr. Bragdon therefore pointed out on remand, as he has all along, that experts on both sides agree the risk to an uninfected dentist working on an HIV-infected patient **is greater than** the risk to an uninfected patient from an infected dentist (87a, 191a, 246a, 570a, 581a). Dr. Bragdon also pointed out this assessment of the relative risks was repeatedly made known to dentists, in professional publications meant for dentists, on or before September 1994 (191a (referring to 1991 first edition of *Practical Infection Control in Dentistry*), 570a, 253a). The Appeals Court ignored this, too, in its supposed determination of what an objectively reasonable dentist could think in 1994.

G. The Appeals Court's Decision So Far Departs From The Accepted And Usual Course Of Judicial Proceedings That The Court Should Exercise Its Supervisory Powers.

To summarize, whatever evidence Dr. Bragdon produced, the Appeals Court ignored. The evidence the Appeals Court took seriously are inadmissible assertions by the Dental Association in an amicus brief and Ms. Abbott's experts' affidavits, the evidentiary bases of which this Court thought were too unclear to justify summary judgment. The public health authority evidence the Appeals Court took most seriously is language in the

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[4] After Dr. Bragdon called the Appeals Court's attention to Dr. Valdiserri's testimony, Dr. Valdiserri notified the Appeals Court that he wants to recant his live testimony, and on reflection he really meant to say "possible" not "probable." *Eppur si muove* (and yet it moves).

1987 Guidelines. The Appeals Court thought summary judgment against Dr. Bragdon was warranted based on language in the 1987 Guidelines. Yet the 1987 Guidelines literally say the opposite of what the Appeals Court says they say. Under these circumstances, it cannot be said the Appeals Court accomplished the goal set by the Court of a fully informed determination. Allowing the Appeals Court's decision to stand would trivialize in the extreme the Court's previous decision to vacate and remand, and it would be a tremendous injustice to Dr. Bragdon who faces the possibility of having to pay Ms. Abbott's attorneys' and experts' fees in excess of $300,000. It would also create in the law an unjustified direct threat double-standard, it would confuse and befuddle courts and parties who deserve real guidance instead, and it would encourage health care providers to practice at safety levels below published minimum guidelines. The Court should therefore exercise its supervisory powers to review and remand this case for a trial.

II. THE APPEALS COURT'S DECISION CREATES A CONFLICT AMONG THE CIRCUITS, AND AN UNJUSTIFIED DOUBLE-STANDARD IN CONFLICT WITH THE COURT'S DECISION, CONCERNING WHAT LEVEL OF RISK IS LEGALLY SIGNIFICANT.

Cases from other circuits have consistently held the risk to patients from HIV-infected health care workers, including dentists, during invasive procedures is legally significant. *Estate of Mauro v. Borgess Med. Ctr*, 137 F.3d 398 (6th Cir. 1998) (surgical technician); *Doe v. University of Md. Medical Sys. Corp.*, 50 F.3d 1261 (4th Cir. 1995) (neurosurgical resident); *Bradley v. University of Tex. M.D. Anderson Cancer Ctr.*, 3 F.3d 922 (5th Cir. 1993) (surgical technician) (per curiam), *cert. denied*, 114 S. Ct. 1071 (1994); *Leckelt v. Board of Comm'rs of Hosp. Dist. No. 1*, 909 F.2d 820 (5th Cir. 1990) (practical nurse); *Scoles v. Mercy Health Corp. of S.E. Pa.*, 887 F. Supp. 765 (E.D. Pa. 1994) (orthopedic surgeon); *Doe v. Washington Univ.*, 780

F. Supp. 628 (E.D. Mo. 1991) (dental student).

Every single expert that has considered the issue on either side further agrees the risk to an uninfected health care worker, including a dentist, when performing invasive procedures on an HIV-infected patient, is equal to and probably greater than the risk to an uninfected patient from an infected worker (191a, 246a, 253a, 570a, 581a). Yet, the Appeals Court maintains Dr. Bragdon does not even raise a genuine issue for trial. Logically, this creates either a direct conflict among the circuits on what level of risk is legally significant, or a double standard whereby dentists in non-emergency situations are compelled by federal law to take substantially greater risks than patients and other citizens.

This double-standard was described in a Harvard Law Review case comment as a "double standard for dealing with HIV infection in the health-care setting," which would

> leave[] the nation in the curious position of having an antidiscrimination statute that declares all individuals with HIV to be equal under the law, but with some "more equal than others."

Comment, *Federal Statutes and Regulations: The Americans with Disabilities Act*, 112 Harv. L. Rev. 283, 293 (Nov. 1998) (citing and quoting Orwell's *Animal Farm*). According to the author, such a double-standard is contrary to the "plain language of the statutory provisions," contrary to legislative history, and "makes little sense from a policy standpoint," *id.* at 291-292. Equally important is that it contradicts the Court's plain holding:

> Efforts to clarify dentists' ethical obligations and to encourage dentists to treat patients with HIV infection with compassion may be commendable, but the question under the statute is one of statistical likelihood, not professional responsibility (24a).

The behavior of the Appeals Court on remand illustrates how,

> [w]ithout a clear and uniform standard, trial or appellate court judges finding no significant risk as a matter of law will undoubtedly be relying more on their own personal perspectives than on articulated legal standards. When, as here, the question can literally be one of life and death, defendants are entitled to the collective perspectives of a jury of their peers rather than the views of a single person—especially if personal perspectives rather than legal standards are being used.

Id. at 293.

In accordance with the logic of Justice Ginsburg's concurrence, Dr. Bragdon agrees that

> unresolved scientific questions [should] be resolved in favor of the uninfected person. Presumably, this same standard would apply whether the infected person was a patient or a provider, and if so, would harmonize *Bragdon* with the "reverse cases."

Id. at 292-93.

The Court should therefore grant certiorari and remand for a trial, thereby eliminating an invidious double-standard and resolving what is otherwise a conflict among circuits on the level of risk legally significant under the ADA.

III. CERTIORARI SHOULD BE GRANTED BECAUSE CASES GENERALLY FAIL TO PROVIDE EMPLOYERS AND PROFESSIONALS ACROSS THE COUNTRY WITH DESERVED GUIDANCE ON WHAT LEVEL OF RISK IS LEGALLY SIGNIFICANT, AND BECAUSE THE APPEALS COURT'S DECISION SO DISTORTS THE MEDICAL AND OBJECTIVE EVIDENCE AVAILABLE TO DENTISTS

IN 1994 THAT NO DENTIST OR OTHER PROFESSIONAL TODAY COULD HOPE TO COMPREHEND HOW THE LAW CALCULATES, OR EXPECTS OTHERS TO CALCULATE, THE SIGNIFICANCE OF A RISK.

A premise of due process in a democratic society is that people are entitled to some reasonable indication of what they must do to conform to the law. This is a reason why the "existence, or nonexistence, of a significant risk must be determined *from the standpoint of the person who refuses the treatment or accommodation*, and the risk assessment must be based on medical or other objective evidence" (21a). However, under current case law, reasonable professionals and employers have no way of knowing what level of risk is significant under the ADA or how to calculate it.

For example, how could Dr. Bragdon know that when CDC guidelines said universal precautions should be the minimum precautions when performing an invasive dental procedure, he should have read that as "maximum precautions"? In the future, how is a doctor or employer supposed to know whether, in the eyes of the law, minimum means minimum or maximum?

How was Dr. Bragdon supposed to calculate the significance of the risk? Was he allowed to rely on, as he did, publicly disclosed CDC calculations of the risk to dental patients and the generally accepted view that the risk of HIV infection to dental workers is as great? (106a-112a) Was he allowed, as he did, to calculate the risk as a scientifically minded person would based on published rates of infection and rates of sharps injuries? (91a-103a) Apparently not, since the Appeals Court rejected without comment such calculations. In the future, how is an objectively reasonable professional or employer supposed to calculate the risk? The "probability that the potential injury will actually occur" is a factor that public accommodations and employers *must* consider under

Title I and Title III regulations when evaluating a potential direct threat. *See* 28 C.F.R. § 36.208(c); 29 C.F.R. § 1630.2(r). If the calculations relied on by Dr. Bragdon are not admissible even to raise a genuine issue of fact, then what are? Public accommodations and employers have the right to some indication from courts as to how they are supposed to figure out if there is a direct threat. [5]

From the standpoint of reasonable dentists in 1994, how were they to know that courts would, without explanation, treat a policy statement of the Dental Association as a higher form of expert opinion, when OSHA concluded, first, that the Dental Association's contentions concerning the risk of HIV to dentists are "confusing and not supported by the data", and second, that other medical evidence is consistent with the conclusion there is a significant HIV risk to dentists? 56 Fed. Reg. at 64092.

Knowledgeable dentists in 1994 also knew the published, objective rate of HIV infection after gloves failed to prevent a percutaneous HIV exposure, was between 0.36% and 0.47% (i.e., between 3 to 5 in a 1000) (94a-98a); and that therefore, *just one* accidental needlestick during an entire lifetime of practice, when working on an HIV positive patient, is a 3 to 5 in a 1000 risk of death,

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[5] *But see*, *Onishea v. Hopper*, 126 F.3d 1323, 1334 (11th Cir. 1997) (remanding for the "quantitative analysis necessary to find a significant risk," but giving no indication how to perform the analysis or what level of quantitative risk is legally significant), *vacated for en banc review*, 133 F.3d 1377 (11th Cir. 1998); *E.E.O.C. v. Union Pacific R.R.*, 6 F. Supp.2d 1135, 1139 (D. Idaho 1998) (stating defendant should have cited "studies or other literature on the subject of the ability of monocular-sighted person to drive safely," but giving no indication what level of risk is significant); *E.E.O.C. v. Exxon Corp*, 1 F. Supp.2d 635, 645 (N.D. Tex. 1998) (stating defendant must show there is a "direct threat," but giving no indication what level of risk is significant or how to show it).

which is many times greater than the level of risk deemed legally significant by OSHA, 56 Fed. Reg. at 64092. This is overwhelming evidence of a direct threat from the standpoint of dentists in 1994.

The point deserves emphasis.

OSHA has established what is an unacceptable lifetime risk for HIV exposure. That risk is 0.1% or 1 in a 1000. Thus, OSHA has established that a single HIV needlestick exposure in a lifetime of practice is unacceptable! So how can Ms. Abbott seriously maintain that HIV needlestick injuries are not significant risks? How could an objectively reasonable dentist in 1994 *not* think there was a significant risk when working on HIV-infected patients, when just one slip during an entire lifetime of practice would put him at between three to five times what OSHA sets as an unacceptable lifetime hazard?

This point was also made clear to the Appeals Court (96a-98a). Based on its decision, however, health care workers again have no way of knowing whether this fact has any relevance to how they are expected to recognize a direct threat. Is OSHA's 1 in a 1000 standard for a "clearly significant risk," which was derived from dicta in *Industrial Union Dep't, AFL-CIO v. American Petroleum Institute*, 448 U.S. 607, 655 (1980), not applicable to a direct threat analysis under the ADA? Or does the ADA expect health care professionals to never have an accidental needlestick during an entire lifetime of practice?

Other reasons why the Court should exercise its supervisory power is that the Appeals Court's opinion is another example of conflicting opinions and a chronic lack of meaningful guidance from courts on the direct threat issue.

For example, the district court in *Nunes v. Wal-Mart Stores, Inc.*, ___ F.3d ___, 1999 WL 33703 (9th Cir. 1999), granted summary judgment to defendant on a direct threat analysis based on the possibility that a cashier might drop a heavy object on a customer during a fainting spell. The

Ninth Circuit reversed and remanded for a trial, acknowledging a genuine issue for trial even though the cashier's doctor testified the risk was "extremely unlikely," and no one has ever heard of such a thing actually happening. Under this standard, Dr. Bragdon is clearly entitled to a trial.

Dr. Bragdon is also entitled to a trial under the standard applied in *Doe v. Dekalb County Sch. Dist.*, 145 F.3d 1441 (1998), where there was a bench trial on the significance of the risk of an HIV-infected teacher being involved in blood-to-blood contact with a student in a class of students with sometimes violent behavioral disorders. Unfortunately, the district court failed, like so many courts, to articulate a meaningful risk analysis, quantitative or qualitative, and relied on "terse findings of fact and conclusions of law." The Eleventh Circuit remanded for *informative* findings and conclusions concerning the level of risk and probabilities involved.

Dr. Bragdon is also entitled to a trial under the standard applied in *Montalvo v. Radcliffe*, ___ F.3d ___, 1999 WL 65624 (4th Cir. 1999), affirming a factual finding at trial that an HIV-infected student poses a direct threat to other students in a combat-oriented martial arts class.

The unscientific, intuitive, non-mathematical, "know it when I see it" summary judgment approach to risk, has also led to inconsistent conclusions. In *Dipol v. New York City Transit Auth.*, 999 F. Supp. 309 (E.D.N.Y. 1998), the court held at summary judgment it was safe (i.e., no significant risk) for a person who had intermittent vision problems and only "fair control" of his diabetes to operate on public tracks in hazardous conditions, whereas in *Daugherty v. City of El Paso*, 56 F.3d 695 (5th Cir. 1995), *cert. denied*, 111 S. Ct. 1263 (1996), the court upheld summary judgment it was unsafe (i.e., a significant risk) for diabetics with similar problems ever to drive city vehicles. In *E.E.O.C. v. Amego Inc.*, 110 F.3d 135 (1st Cir. 1997), the court upheld summary judgment it was unsafe (i.e., a

significant risk) for an employee who had been suicidal to continue administering drugs to seriously disabled persons, even though the employee's own physician and a social worker saw no difficulty with her continuing to do that job, whereas in *E.E.O.C. v. Union Pacific R.R.*, 6 F. Supp.2d 1135 (D. Idaho 1998), the court granted summary judgment that it was safe (i.e., no significant risk) for monocular-sighted persons without depth perception to drive safely around switching yards.[6]

The real lesson from these cases, besides their inconsistency and lack of statistical analysis, is they should not have been decided by summary judgment. Given that

> "neither Congress nor the Supreme Court has informed us what the norm is" in determining whether a given level of risk is significant or not, it is questionable whether it is ever appropriate to grant summary judgment when, as in *Bragdon*, reasonable minds could differ. In *Bragdon*, the Justices differed substantially on whether the risk was significant, but none of them explained what a "significant" risk is, which implies that they only "know it when [they] see it." Without a clear and uniform standard, trial court judges

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[6] Other examples of the intuitive, unscientific, "know it when I see it" summary judgment approach to direct threat are *LaChance v. Duffy's Draft House, Inc.*, 146 F.3d 832 (11th Cir. 1998) (upheld summary judgment of direct threat posed by line cook, even though he later worked for 13 months without incident, rarely had seizures, and seizures consisted of momentary disorientation); *Burroughs v. City of Springfield*, 163 F.3d 505 (8th Cir. 1998) (upheld summary judgment of direct threat posed by diabetic police officer when testimony was merely he "could conceivably be dangerous to the public"); *Turco v. Hoechst Celanese Corp.*, 101 F.3d 1090 (5th Cir. 1996) (diabetic chemical process worker described with no medical or statistical support as "walking time bomb").

> finding no significant risk as a matter of law will undoubtedly by relying more on their own personal perspectives than on articulated legal standards. When, as [in *Bragdon*], the question can literally be one of life and death, defendants are entitled to the collective perspectives of a jury of their peers.

Comment, *supra*, 112 Harv. L. Rev. at 293.

By granting certiorari and remanding for a trial, the Court can and should send a clear message that "direct threat" is first and foremost an issue for full exploration at trial, and rarely, if ever, should it be decided by summary judgment. This will put an end to the alarming and mystifying tendency of federal courts to think they must decide by summary judgment whether a risk is significant. These issues are properly the subject of in-depth analysis at trial.

IV. CERTIORARI SHOULD BE GRANTED BECAUSE THE APPEALS COURT'S DECISION ENCOURAGES HEALTH CARE WORKERS TO PRACTICE BELOW MINIMUM SAFETY STANDARDS.

The 1987 Guidelines specifically say universal precautions should be the minimum precautions for all invasive procedures (459a). The 1993 Guidelines carry that principle forward by stating the principles of infection control remain unchanged (528a), by using the concepts of universal infection control strategies recommended for all dental settings versus specific infection control strategies, and by indicating that although a set of universal precautions common to all dental-treatment settings "should reduce the risk," the "unique nature of most dental procedures . . . also may require specific [i.e., non-universal, additional] strategies directed to the prevention of transmission of pathogens" (528a; *see* 128a-130a).

Now the Appeals Court has transformed CDC 1987 voluntary minimum guidelines into mandatory ceilings on levels of safe care, and ruled that anyone who practices

above the minimum safety standards is a civil rights violator. Such a ruling is unprecedented in the history of law and medicine. Never before has a doctor or an employer been punished for wanting to practice too safely, for putting too high a value on human life. In view of the fact that OSHA's 1991 regulations required Maine dentists to treat everyone, including children, as though they might be infected with HIV, when over 99% of them are not infected, it is mind boggling to say that a dentist was "too careful" in treating a known HIV positive patient.

V. THE APPEALS COURT'S DECISION SHOULD BE REVIEWED BECAUSE IT CONFLICTS WITH ESTABLISHED NORMS OF SAFETY IN OTHER FIELDS.

Dr. Bragdon introduced admissible, reliable scientific evidence that the mortal danger to him was on the order of 1 in 67,000 per injection, and he provided a reliable, scientific analysis as to how and why this level of risk is consistent with observed occupational transmissions of HIV (91a-119a, 147a-151a). The Appeals Court ruling entails that this level of risk is insignificant to dentists as a matter of law. Yet, for example, when it comes to airline safety, traffic safety, or food safety, no one would tolerate that level of risk or consider it insignificant. If 1 out of every 100,000 planes crashed, or hamburgers were poisonous, or gasoline tanks exploded, there would be a huge outcry. Likewise, if Dr. Bragdon compelled one of his employees to face the risk of HIV needlestick, he could be sued successfully under the Occupational Safety and Health Act, 29 U.S.C. § 654(a)(1), on the ground he knew specific OSHA regulations did not purport to eliminate a significant risk of HIV infection from such injuries. *See International Union U.A.W. v. General Dynamics Land Sys. Div.*, 815 F.2d 1570, 1577 (D.C. Cir. 1987) (employer liable under general duty clause despite compliance with specific regulations when employer knows specific standards will not protect against a particular hazard).

So why should a 1 in 67,000 or greater risk of death be legally insignificant for dentists? It should not, but the psychology, rather than the science of risk, may be playing a decisive role in the case. Specifically, the vast majority of people in the United States (over 99%) do not have HIV (440a), so for billions of routine dental procedures there is no HIV issue. This means HIV-infected dental workers are far, far less common than dead people would be if 1 in every 100,000 hamburgers were poisoned. The problem seems invisible. For the relatively rare dental procedure (substantially less than 1%) where an HIV risk is present, dentists become infected in only a very small fraction of those cases, and typically by patients who did not reveal their HIV status, or who did not know it. If the dentist is gay, no one will believe he was infected on the job. If the dentist was already infected, no one tests if he was re-infected at work. If the dentist is infected, he probably will not know for quite a while, perhaps years, and when he does, he probably will not make it public since it will destroy his practice.

Dentists suffer many needlestick and other sharps injuries each year, but do not stop every time to find out if the patient is HIV positive or take an HIV test. No law says a patient must agree to testing. Even if the patient is HIV positive, there is little a dentist can do unless he is willing to run to a hospital to start a brutal course of post exposure prophylaxis (195a-197a). Human nature is to move on and hope for the best, knowing few patients have HIV, and even if this one does, odds are only about 4 in a 1000 of becoming infected. Many dentists are willing to face those odds, and many public health officials see no practical alternative. Dr. Bragdon was willing to face the risk for the sake of Ms. Abbott, but he wanted to be as careful as possible for himself, his assistants, and his patient. For that he is branded a civil rights violator and now faces legal exposure in excess of $300,000.

VI. THE COURT SHOULD ADDRESS THE RECURRING ISSUE OF BURDEN OF PROOF UNDER TITLE III OF THE ADA.

On remand Dr. Bragdon raised again the issue of who has the burden of proof on direct threat (163a-166a). With respect to the risk of aerosol transmission of HIV (158a-163a), and other risks in cases where there are no definitive studies to guide a risk analysis, *e.g.*, *Onishea*, 126 F.3d 1323; *Amego Inc.*, 110 F.3d 135; *Union Pacific R.R.*, 6 F. Supp.2d at 1139; *Exxon Corp*, 1 F. Supp.2d at 645, the assignment of the burden of proof essentially determines the outcome of the case. Prospectively, it determines whether public accommodations may lawfully act in conformity with sound practical reason in the absence of definitive scientific proof (162a-163a), or whether, if they do, they will be liable for disability discrimination. Sound policy is to err, "if at all, on the side of caution" (Ginsburg, J., concurring) (28a). The Court should therefore grant certiorari to review who has the burden of proof on direct threat.

CONCLUSION

Four members of the Court already believe this case is wrongly decided. Three were uncertain, having serious doubts as to the Appeals Court's interpretation of the 1993 Guidelines. Did the 1993 Guidelines inform dentists there is no significant risk? It does not seem so. But perhaps somewhere else in some other source available before September 1994, there is language justifying summary judgment against Dr. Bragdon.

For five years Dr. Bragdon has said there is none.

Is there any doubt that if there were any such language supporting Ms. Abbott, it would have been plastered all over her briefs, especially given the resources she brought to the case, e.g., the American Medical Association, CDC, the Department of Justice, and the State of Maine?

On remand, the most Ms. Abbott and the Appeals Court could find to justify summary judgment against

Dr. Bragdon was unidentified language in the 1987 Guidelines. This is more obviously wrong than before, since the 1987 Guidelines literally say the opposite of what the Appeals Court says they say.

This really is a very simple case for summary judgment purposes. *See, e.g.,* 120a n.12. No public health authority publication available in September 1994 warrants summary judgment for Ms. Abbott. The Dental Association's 1991 Policy is not admissible expert testimony, or if it is, it carries no more weight than any other *ipse dixit* expert opinion. Dr. Kuvin's expert testimony on behalf of Dr. Bragdon (e.g., 573a-574a, 602a-610a), and the other sources of medical and objective information presented by Dr. Bragdon, raise a genuine issue of fact that an objectively reasonable dentist could have and would have thought there was a direct threat based on information available in September 1994.

Dr. Bragdon knows it is uncommon, but not unprecedented, for the Court to grant certiorari because a case is wrongly decided. *See* Robert L. Stern, et al., *Supreme Court Practice* § 4.17 (7th ed. 1993). This is the unusual situation when there are national issues at stake and the Court can also do justice in the individual case.

With respect to the four members of the Court who would have already remanded for trial, Dr. Bragdon wonders what purpose would be served by declining to grant certiorari, when the justices are already on record as favoring a trial and three of their colleagues were uncertain. Certainly the purposes of blind efficiency and maintaining a tradition whereby the Court rarely reviews anything but pure issues of law would be served by denying certiorari. Just as certainly there is no danger the floodgates to the Court will open, as this is an extremely unusual situation, and at all times the Court has the discretion to regulate its docket. Nor should the Court overlook the real need, nationally, for guidance on the direct threat issue. Simply by ordering a remand for trial, and expressing a strong

preference for trials in such cases, the Court can effectively wipe out an unjustified legal double-standard and conflict among the circuits created by the Appeals Court's decision.

With respect to the three members of the Court who so doubted the propriety of summary judgment as to vacate and remand to the Appeals Court, Dr. Bragdon wonders what purpose would be served by now letting stand a decision on remand that is more clearly flawed than the first. The goals already set by the Court of a full exploration of the facts and a fully informed decision should be attained by granting certiorari and remanding for a trial.

Finally, though the Court may always prefer regular briefing and oral argument following a grant of certiorari, it might also consider asking, in the certiorari petition review process itself, that Ms. Abbott oppose certiorari by explaining why the Court should not grant certiorari, summarily vacate and remand for trial. The relevant material is contained in this petition and the petition appendix (e.g., 62a-169a). If five or more members of the Court agree there is a genuine issue for trial, a short decision stating a preference for trials in such cases will work justice in the individual case, will eliminate an unjustified direct threat double-standard in the law, resolve unnecessary conflict among the circuits on what level of risk is legally significant under the ADA, and foretell an end to the intuitive, unscientific, ad hoc, "I know it when I see it" summary judgment approach to direct threat.

Respectfully Submitted,

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Sidney ABBOTT, et al., Plaintiffs, Appellees,

v.

Randon BRAGDON, D.M.D., Defendant, Appellant.

No. 96-1643.

United States Court of Appeals,
First Circuit.

Reheard Dec. 9, 1998.

Dec. 29, 1998.

Patient infected with human immunodeficiency virus (HIV) brought action under Americans with Disabilities Act (ADA) and Maine Human Rights Act against dentist who refused to treat her in his office. The United States District Court for the District of Maine, 912 F.Supp. 580, granted summary judgment for patient, and dentist appealed. The Court of Appeals, 107 F.3d 934, affirmed. Dentist petitioned for certiorari. The United States Supreme Court, 118 S.Ct. 2196, affirmed in substantial part, but remanded with instructions. On remand, the Court of Appeals, Selya, Circuit Judge, held that dentist's performance of cavity-filling procedure on patient did not pose "direct threat" to others.

Affirmed.

CIVIL RIGHTS k107(4)
78k107(4)
Dentist's performance of cavity-filling procedure on patient with asymptomatic human immunodeficiency virus (HIV) did not pose "direct threat" to others, within exception to ADA's prohibition against discrimination, considering "universal precautions" prescribed in Dentistry Guidelines formulated by Centers for Disease Control (CDC), and Policy on acquired immune deficiency syndrome (AIDS), HIV Infection and Practice of Dentistry propounded by American Dental Association. Americans with Disabilities Act of 1990, § 302, 42 U.S.C.A. § 12182.

See publication Words and Phrases for other judicial constructions and definitions.

CIVIL RIGHTS k119.1
78k119.1
Dentist's performance of cavity-filling procedure on patient with asymptomatic human immunodeficiency virus (HIV) did not pose "direct threat" to others, within exception to ADA's prohibition against discrimination, considering "universal precautions" prescribed in Dentistry Guidelines formulated by Centers for Disease Control (CDC), and Policy on acquired immune deficiency syndrome (AIDS), HIV Infection and Practice of Dentistry propounded by American Dental Association. Americans with Disabilities Act of 1990, § 302, 42 U.S.C.A. § 12182.
See publication Words and Phrases for other judicial constructions and definitions.

***87** John W. McCarthy, with whom Brent A. Singer and Rudman & Winchell, LLC, Bangor, ME, were on brief, for defendant.

Stephen C. Whiting and the Whiting Law Firm, P.A., Portland, ME, on brief for Cary Savitch, M.D., amicus curiae.

Scott Somerville on brief, for Dentists for Preservation of Professional Judgment, amicus curiae.

Robert J. Masini and Diver, Grach, Quade & Masini, Waukegan, IL, on brief for American Association of Forensic Dentists, amicus curiae.

Richard L. Hill, Lance N. Long and Hill, Johnson & Schmutz P.C., on brief for Clinical Research Associates, amicus curiae.

Bennett H. Klein, with whom Gay and Lesbian Advocates & Defenders, David G. Webbert, Johnson & Webbert, LLP, Augusta, ME, Wendy E. Parmet, Boston, MA, were on brief, for

plaintiff Sidney Abbott.

John E. Carnes, Commission Counsel, on consolidated brief for intervenor- plaintiff Maine Human Rights Commission.

Thomas E. Chandler, Attorney, U.S. Dept. of Justice, Washington, DC, with whom Bill Lann Lee, Acting Assistant Attorney General, and Jessica Dunsay Silver, Washington, DC, were on brief, for United States of America, amicus curiae.

Peter M. Sfikas, Chicago, IL, Mark S. Rubin, Kathleen Todd, Jill A. Wolowitz, Scott M. Mendel, Chicago, IL, Bell, Boyd & Lloyd and Patrick J. Quinlan, Providence, RI, on brief for American Dental Ass'n, amicus curiae.

Before SELYA, Circuit Judge, CYR, Senior Circuit Judge, and STAHL, Circuit Judge.

***88** SELYA, Circuit Judge.

This case involves a claim of disability-based discrimination brought by an asymptomatic HIV-positive individual, Sidney Abbott, against Randon Bragdon, a dentist who refused to fill Ms. Abbott's cavity in his office. The district court found Ms. Abbott's case compelling and granted summary judgment in her favor. See Abbott v. Bragdon, 912 F.Supp. 580 (D.Me.1995) (Abbott I). We affirmed, albeit on somewhat different reasoning. See Abbott v. Bragdon, 107 F.3d 934 (1st Cir.1997) (Abbott II). The Supreme Court affirmed our decision in substantial part, but remanded with instructions that we reexamine several pieces of evidence. See Bragdon v. Abbott, --- U.S. ----, 118 S.Ct. 2196, 141 L.Ed.2d 540 (1998) (Abbott III). We ordered supplemental briefing, entertained a new round of oral argument, and now reaffirm the district court's entry of summary judgment.

I

We limned the pertinent facts in our earlier opinion, see Abbott II, 107 F.3d at 937-38, and it would be pleonastic to rehearse them here. To lend context, it suffices to remind the reader that Ms. Abbott, who was infected with the Human Immunodeficiency Virus (HIV), went to Dr. Bragdon's Bangor, Maine office for a dental appointment in September 1994; that she was then in the asymptomatic phase of the disease and so informed the dentist; and that, after Dr. Bragdon discovered a cavity, he refused to fill it in his office. Ms. Abbott sued, claiming violations of the Americans With Disabilities Act (the ADA), 42 U.S.C. § 12182 (1994), and the Maine Human Rights Act, 5 Me.Rev.Stat. Ann. tit. 5, § 4592 (West Supp.1998). [FN1]

FN1. Throughout this litigation, the asserted violations of the Maine statute have been treated as following the ADA analysis precisely. See Abbott II, 107 F.3d at 937 n. 1. Accordingly, we need not address the Maine statute further.

The earlier phases of this litigation established that asymptomatic HIV constitutes a disability under the ADA. See Abbott III, --- U.S. at ----, 118 S.Ct. at 2207 (aff'g Abbott II, 107 F.3d at 942). The sole remaining question is whether performance of the cavity-filling procedure posed a "direct threat" to others and thereby came within an exception to the ADA's broad prohibition against discrimination. See Abbott II, 107 F.3d at 943; see also 42 U.S.C. § 12182(b)(3) (stating the exception and defining a direct threat under the ADA as "a significant risk to the health or safety of others that cannot be eliminated by a modification of policies, practices, or procedures or by the provision of auxiliary aids or services").

In the earlier appeal, our rejection of Dr. Bragdon's direct threat defense relied in part on our reading of (i) the 1993 Dentistry Guidelines (the Guidelines) formulated by the Centers for Disease Control (CDC), and (ii) the Policy on AIDS, HIV Infection and the Practice of Dentistry (the Policy) propounded

by the American Dental Association (the Association). See Abbott II, 107 F.3d at 945-46. Each of these documents indicated to us that the use of so-called "universal precautions" would render the risk of performing the cavity-filling procedure in a dental office insignificant. See id. We also noted the absence of a trialworthy showing by Dr. Bragdon as to any direct threat. See id. at 946-48. The Supreme Court remanded to permit a reevaluation of the evidence on this issue, and, in particular, a reexamination of the Guidelines and the Policy. See Abbott III, 118 S.Ct. at 2211-13. In doing so, the Court took pains to explain that its disposition did not debar us from again reaching the same result. See id. at 2213.

II

In compliance with the Court's directive, we have reexamined the evidence to determine whether summary judgment was warranted. In order to reverse our course, we would have to find, contrary to our original intuition, either that (i) Ms. Abbott did not merit judgment as a matter of law even in the absence of disputed facts, or (ii) that Dr. Bragdon had submitted sufficient evidence to create a genuine issue of material fact as to his direct threat defense. In our reexamination, we apply conventional summary judgment jurisprudence, drawing all reasonable *89 factual inferences in favor of Dr. Bragdon (as the party opposing brevis disposition). See Abbott II, 107 F.3d at 938 (citing Smith v. F.W. Morse & Co., 76 F.3d 413, 428 (1st Cir.1996)). Despite the leniency of this approach, we do not indulge "conclusory allegations, improbable inferences, and unsupported speculation." Medina- Munoz v. R.J. Reynolds Tobacco Co., 896 F.2d 5, 8 (1st Cir.1990).

A. Ms. Abbott's Evidence.

The Supreme Court raised questions regarding whether the Guidelines, which state that use of the universal precautions therein described "should reduce the risk of disease transmission in the dental environment," necessarily imply that the reduction

of risk would be to a level below that required to show direct threat. Abbott III, 118 S.Ct. at 2211 (quoting Guidelines). We have reconsidered this point.

The CDC did not write the 1993 Guidelines in a vacuum, but, rather, updated earlier versions issued in 1986 and 1987, respectively. The 1986 text calls the universal precautions "effective for preventing hepatitis B, acquired immunodeficiency syndrome, and other infectious diseases caused by bloodborne viruses." The 1987 edition explains that use of the universal precautions eliminates the need for additional precautions that the CDC formerly had advocated for handling blood and other bodily fluids known or suspected to be infected with bloodborne pathogens. Neither the parties nor any of the amici have suggested that the 1993 rewrite was intended to retreat from these earlier risk assessments, and we find no support for such a position in the Guidelines' text. Thus, we have again determined that the Guidelines are competent evidence that public health authorities considered treatment of the kind that Ms. Abbott required to be safe, if undertaken using universal precautions.

Second, the Court questioned the appropriate weight to accord the Policy, expressing concern that the Policy might be based in whole or in part on the Association's view of dentists' ethical obligations, rather than on a pure scientific assessment. See Abbott III, 118 S.Ct. at 2211-12. The supplemental briefing that we requested yielded a cornucopia of information regarding the process by which the Policy was assembled. We briefly recount the undisputed facts.

The Association formulates scientific and ethical policies by separate procedures, drawing on different member groups and different staff complements. The Association's Council on Scientific Affairs, comprised of 17 dentists (most of whom hold advanced dentistry degrees), together with a staff of over 20 professional experts and consultants, drafted the Policy at issue here. By contrast, ethical policies are drafted by the Council on Ethics, a wholly separate body. Although the Association's

House of Delegates must approve policies drafted by either council, we think that the origins of the Policy satisfy any doubts regarding its scientific foundation.

For these reasons, we are confident that we appropriately relied on the Guidelines and the Policy. Moreover, as the Supreme Court acknowledged, see id. at 2212, these two pieces of evidence represent only a fraction of the proof advanced to support Ms. Abbott's motion. For example, she proffered the opinions of several prominent experts to the effect that, in 1994, the cavity- filling procedure could have been performed safely in a private dental office, as well as proof that no public health authority theretofore had issued warnings to health care providers disfavoring this type of treatment for asymptomatic HIV-positive patients. These materials, in and of themselves, likely suffice to prove Ms. Abbott's point. Thus, we again conclude, after due reevaluation, that Ms. Abbott served a properly documented motion for summary judgment.

B. Dr. Bragdon's Evidence.

We next reconsider whether Dr. Bragdon offered sufficient proof of direct threat to create a genuine issue of material fact and thus avoid the entry of summary judgment. In Abbott II, we canvassed eight items of evidence adduced by Dr. Bragdon in an effort to demonstrate a genuine issue of material fact. See Abbott II, 107 F.3d at 946-48. The Supreme Court suggested that one such piece of evidence--the seven cases that the CDC considered "possible" HIV patient-to-*90 dental worker transmissions--should be reexamined. See Abbott III, 118 S.Ct. at 2212.

The Court's concern revolved around how the word "possible" was understood in this context at the relevant time. To frame the issue, the Court noted that the CDC marks an HIV case as a "possible" occupational transmission if a stricken worker, who had no other demonstrated opportunity for infection, simply failed to present himself for testing after being exposed to the

virus at work. See id. The Court speculated that if this definition of "possible" was not available in September 1994, the existence of seven "possible" cases "might have provided some, albeit not necessarily sufficient, support for [Dr. Bragdon's] position." Id. In other words, if a dentist knew of seven "possible" occupational transmissions to dental workers without understanding that "possible" meant no more than that the CDC could not determine whether workers were infected occupationally, he might reasonably regard the risk of treating an HIV-infected patient to be significant.

Upon reexamination of the record, we find that the CDC's definition of the word "possible," as used here, had been made public during the relevant period. The record contains two scientific articles published before Ms. Abbott entered Dr. Bragdon's office which explained this definition. See Louise J. Short & David M. Bell, Risk of Occupational Infection With BloodBorne Pathogens in Operating and Delivery Room Settings, 21 Am. J. Infection Control 343, 345 (1993); John A. Molinari, HIV, Health Care Workers and Patients: How to Ensure Safety in the Dental Office, 124 J. Am. Dental Ass'n 51, 51-52 (1993). Since an objective standard pertains here, see Abbott III, 118 S.Ct. at 2211; Abbott II, 107 F.3d at 944, the existence of the list of seven "possible" cases does not create a genuine issue of material fact as to direct threat.

In his supplemental briefing and oral argument, Dr. Bragdon has drawn our attention again to the CDC's report of 42 documented cases of occupational transmission of HIV to health-care workers (none of whom were dental workers). He repeats his argument that, because dental workers are subject to dangers similar to those faced by other health-care workers, these cases can be extrapolated to create an issue of fact as to the degree of risk to dental workers in September 1994. We previously held that this evidence was insufficient without a documented showing that the risks to dentists and other health-care workers are comparable, see Abbott II, 107 F.3d at 947, and the appellant offers us no cogent reason to change our view. The Supreme Court did not

question our position on this front, and Dr. Bragdon points to no record support that we previously might have overlooked.

Our assessment of Dr. Bragdon's, and his amici's, other reprised arguments similarly remains unchanged. Each piece of evidence to which they direct us is still "too speculative or too tangential (or, in some instances, both) to create a genuine issue of material fact." Id. at 948.

III

We need go no further. Upon reflection, we again find that Dr. Bragdon did not submit evidence to the district court demonstrating a genuine issue of material fact on the direct threat issue. Absent such a showing, the district court appropriately entered summary judgment in favor of Ms. Abbott. In espousing that view, we emphasize the case-specific nature of our determination. Our disposition is confined to the facts of record here (as they were presented in the nisi prius court). The state of scientific knowledge concerning this disease is evolving, and we caution future courts to consider carefully whether future litigants have been able, through scientific advances, more complete research, or special circumstances, to present facts and arguments warranting a different decision.

Affirmed.

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Plaintiffs, Appellees,

v.

RANDON BRAGDON, D.M.D.,
Defendant, Appellant.

JUDGMENT

Entered: December 29, 1998

This cause came on to be heard on remand of the Supreme Court of the United States, and was argued by counsel.

Upon consideration whereof, it is now here ordered, adjudged and decreed as follows: The judgment of the district court is affirmed.

By The Court:

PHOEBE MORSE,
CLERK

By: JANICE M. O'NEIL
Chief Deputy Clerk

Randon BRAGDON, Petitioner,
v.
Sidney ABBOTT et al.

No. 97-156.

Supreme Court of the United States

Argued March 30, 1998.

Decided June 25, 1998

Patient infected with the human immunodeficiency virus (HIV) brought action under the Americans with Disabilities Act (ADA) against dentist who refused to treat her in his office. The United States District Court for the District of Maine, 912 F.Supp. 580, granted summary judgment in favor of patient, and dentist-appealed. The First Circuit Court of Appeals, 107 F.3d 934, affirmed. Dentist petitioned for certiorari. The Supreme Court, Justice Kennedy, held that: (1) HIV infection is a "disability" under the ADA, even when the infection has not yet progressed to the so-called symptomatic phase, as a physical impairment which substantially limits the major life activity of reproduction, and (2) with regard to "direct threat" provision of the ADA, the existence, or nonexistence of a significant health risk from treatment or accommodation of a disabled person must be determined from standpoint of the person who refused the treatment or accommodation, but the risk assessment must be based on medical or other objective evidence, and not simply on that person's good-faith belief that a significant risk existed.

Vacated and remanded.

Justice Stevens filed concurring opinion in which Justice Breyer, joined.

Justice Ginsburg filed concurring opinion.

Chief Justice Rehnquist filed opinion concurring in the judgment in part and dissenting in part, in which Justices Scalia and Thomas joined, and in Part II of which Justice O'Connor joined.

Justice O'Connor filed opinion concurring in the judgment in part and dissenting in part.

[West headnotes omitted]

***2198** Syllabus [FN*]

FN* The syllabus constitutes no part of the opinion of the Court but has been prepared by the Reporter of Decisions for the convenience of the reader. See United States v. Detroit Timber & Lumber Co., 200 U.S. 321, 337, 26 S.Ct. 282, 287, 50 L.Ed. 499.

Respondent is infected with the human immunodeficiency virus (HIV), but had not manifested its most serious symptoms when the incidents in question occurred. At that time, she went to petitioner's office for a dental examination and disclosed her HIV infection. Petitioner discovered a cavity and informed respondent of his policy against filling cavities of HIV-infected patients in his office. He offered to perform the work at a hospital at no extra charge, though respondent would have to pay for use of the hospital's facilities. She declined and filed suit under, inter alia, the Americans with Disabilities Act of 1990(ADA), which prohibits discrimination against any individual "on the basis of disability in the ... enjoyment of the ... services ... of any place of public accommodation by any person who ... operates [such] a place," 42 U.S.C. § 12182(a), but qualifies the prohibition by providing: "Nothing [herein] shall require an entity to permit an individual to participate in or benefit from the ... accommodations of such entity where such individual poses a direct threat to the health or safety of others," § 12182(b)(3). The District Court granted respondent summary judgment. The First Circuit affirmed, agreeing with the lower

court that respondent's HIV was a disability under the ADA even though her infection had not yet progressed to the symptomatic stage, and that treating her in petitioner's office would not have posed a direct threat to the health and safety of others. In making the latter ruling, the court relied on the 1993 Dentistry Guidelines of the Centers for Disease Control and Prevention (CDC) and on the 1991 American Dental Association Policy on HIV.

Held:

1. Even though respondent's HIV infection had not progressed to the so-called symptomatic phase, it was a "disability" under § 12102(2)(A), that is, "a physical ... impairment that substantially limits one or more of [an individual's] major life activities." Pp. 2201-2210.

(a) The ADA definition is drawn almost verbatim from definitions applicable to § 504 of the Rehabilitation Act of 1973 and another federal statute. Because the ADA expressly provides that "nothing [herein] shall be construed to apply a lesser standard than ... under ... the Rehabilitation Act ... or the regulations issued ... pursuant to [it]," § 12201(a), this Court must construe the ADA to grant at least as much protection as the regulations implementing the Rehabilitation Act. Pp. 2201-2202.

(b) From the moment of infection and throughout every stage of the disease, HIV infection satisfies the statutory and regulatory definition of a "physical impairment." Applicable Rehabilitation Act regulations define "physical or mental impairment" to mean "any physiological disorder or condition ... affecting ... the ... body['s] ... hemic and lymphatic [systems]." HIV infection falls *2199 well within that definition. The medical literature reveals that the disease follows a predictable and unalterable course from infection to inevitable death. It causes immediate abnormalities in a person's blood, and the infected person's white cell count continues to drop throughout the course of the disease,

even during the intermediate stage when its attack is concentrated in the lymph nodes. Thus, HIV infection must be regarded as a physiological disorder with an immediate, constant, and detrimental effect on the hemic and lymphatic systems. Pp. 2201-2205.

(c) The life activity upon which respondent relies, her ability to reproduce and to bear children, constitutes a "major life activity" under the ADA. The plain meaning of the word "major" denotes comparative importance and suggests that the touchstone is an activity's significance. Reproduction and the sexual dynamics surrounding it are central to the life process itself. Petitioner's claim that Congress intended the ADA only to cover those aspects of a person's life that have a public, economic, or daily character founders on the statutory language. Nothing in the definition suggests that activities without such a dimension may somehow be regarded as so unimportant or insignificant as not to be "major." This interpretation is confirmed by the Rehabilitation Act regulations, which provide an illustrative, nonexhaustive list of major life activities. Inclusion on that list of activities such as caring for one's self, performing manual tasks, working, and learning belies the suggestion that a task must have a public or economic character. On the contrary, the regulations support the inclusion of reproduction, which could not be regarded as any less important than working and learning. Pp. 2205- 2206.

(d) Respondent's HIV infection "substantially limits" her major life activity within the ADA's meaning. Although the Rehabilitation Act regulations provide little guidance in this regard, the Court's evaluation of the medical evidence demonstrates that an HIV-infected woman's ability to reproduce is substantially limited in two independent ways: If she tries to conceive a child, (1) she imposes on her male partner a statistically significant risk of becoming infected; and (2) she risks infecting her child during gestation and childbirth, i.e., perinatal transmission. Evidence suggesting that antiretroviral therapy can lower the risk of perinatal transmission to about 8%,

even if relevant, does not avail petitioner because it cannot be said as a matter of law that an 8% risk of transmitting a dread and fatal disease to one's child does not represent a substantial limitation on reproduction. The decision to reproduce carries economic and legal consequences as well. There are added costs for antiretroviral therapy, supplemental insurance, and long-term health care for the child who must be examined and treated. Some state laws, moreover, forbid HIV-infected persons from having sex with others, regardless of consent. In the context of reviewing summary judgment, the Court must take as true respondent's unchallenged testimony that her HIV infection controlled her decision not to have a child. Pp. 2206-2207.

(e) The uniform body of administrative and judicial precedent interpreting similar language in the Rehabilitation Act confirms the Court's holding. Every agency and court to consider the issue under the Rehabilitation Act has found statutory coverage for persons with asymptomatic HIV. The uniformity of that precedent is significant. When administrative and judicial interpretations have settled the meaning of an existing statutory provision, repetition of the same language in a new statute indicates, as a general matter, Congress' intent to incorporate such interpretations as well. See, e.g., Lorillard v. Pons, 434 U.S. 575, 580-581, 98 S.Ct. 866, 869-870, 55 L.Ed.2d 40. Pp. 2207-2209.

(f) The Court's holding is further reinforced by the guidance issued by the Justice Department and other agencies authorized to administer the ADA, which supports the conclusion that persons with asymptomatic HIV fall within the ADA's definition of disability. The views of agencies charged with implementing a statute are entitled to deference. See Chevron U.S.A. Inc. v. Natural Resources Defense Council, Inc., 467 U.S. 837, 844, 104 S.Ct. 2778, 2782-2783, 81 L.Ed.2d 694. Pp. 2209-2210.

*2200 2. In affirming the summary judgment, the First Circuit did not cite sufficient material in the record to determine, as a matter of law, that respondent's HIV infection posed no direct

threat to the health and safety of others. The ADA's direct threat provision, § 12182(b)(3), stems from School Bd. of Nassau Cty. v. Arline, 480 U.S. 273, 287, 107 S.Ct. 1123, 1130-1131, 94 L.Ed.2d 307, in which this Court reconciled competing interests in prohibiting discrimination and preventing the spread of disease by construing the Rehabilitation Act not to require the hiring of a person who posed "a significant risk of communicating an infectious disease to others," id., at 287, and n. 16, 107 S.Ct., at 1130-1131, and n. 16. The existence of a significant risk is determined from the standpoint of the health care professional who refuses treatment or accommodation, and the risk assessment is based on the medical or other objective, scientific evidence available to him and his profession, not simply on his good-faith belief that a significant risk existed. See id., at 288, 107 S.Ct., at 1131; id., at 288, n. 18, 107 S.Ct., at 1131 n. 18, distinguished. For the most part, the First Circuit followed the proper standard and conducted a thorough review of the evidence. However, it might have mistakenly relied on the 1993 CDC Dentistry Guidelines, which recommend certain universal precautions to combat the risk of HIV transmission in the dental environment, but do not actually assess the level of such risk, and on the 1991 American Dental Association Policy on HIV, which is the work of a professional organization, not a public health authority, and which does not reveal the extent to which it was based on the Association's assessment of dentists' ethical and professional duties, rather than scientific assessments. Other evidence in the record might support affirmance of the trial court's ruling, and there are reasons to doubt whether petitioner advanced evidence sufficient to raise a triable issue of fact on the significance of the risk, but this Court's evaluation is constrained by the fact that it has not had briefs and arguments directed to the entire record. A remand will permit a full exploration of the issues through the adversary process. Pp. 2210-2213.

107 F.3d 934, vacated and remanded.

KENNEDY, J., delivered the opinion of the Court, in which STEVENS, SOUTER, GINSBURG, and BREYER, JJ., joined. STEVENS, J., filed a concurring opinion, in which BREYER, J., joined. GINSBURG, J., filed a concurring opinion. REHNQUIST, C. J., filed an opinion concurring in the judgment in part and dissenting in part, in which SCALIA and THOMAS, JJ., joined, and in Part II of which O'CONNOR, J., joined. O'CONNOR, J., filed an opinion concurring in the judgment in part and dissenting in part.

John W. McCarthy, Bangor, ME, for petitioner.

Bennett H. Klein, for respondent.

Lawrence G. Wallace, Washington, DC, for United States as amicus curiae by special leave of the Court.

Justice KENNEDY delivered the opinion of the Court.

We address in this case the application of the Americans with Disabilities Act of 1990(ADA), 104 Stat. 327, 42 U.S.C. § 12101 et seq., to persons infected with the human immunodeficiency virus (HIV). We granted certiorari to review, first, whether HIV infection is a disability under the ADA when the infection has not yet progressed to the so-called symptomatic phase; and, second, whether the Court of Appeals, in affirming a grant of summary judgment, cited sufficient material in the record to determine, as a matter of law, that respondent's infection with HIV posed no direct threat to the health and safety of her treating dentist.

I

Respondent Sidney Abbott has been infected with HIV since 1986. When the incidents *2201 we recite occurred, her infection had not manifested its most serious symptoms. On September 16, 1994, she went to the office of petitioner Randon Bragdon in Bangor, Maine, for a dental appointment. She

disclosed her HIV infection on the patient registration form. Petitioner completed a dental examination, discovered a cavity, and informed respondent of his policy against filling cavities of HIV-infected patients. He offered to perform the work at a hospital with no added fee for his services, though respondent would be responsible for the cost of using the hospital's facilities. Respondent declined.

Respondent sued petitioner under state law and § 302 of the ADA, 104 Stat. 355, 42 U.S.C. § 12182, alleging discrimination on the basis of her disability. The state law claims are not before us. Section 302 of the ADA provides:

"No individual shall be discriminated against on the basis of disability in the full and equal enjoyment of the goods, services, facilities, privileges, advantages, or accommodations of any place of public accommodation by any person who ... operates a place of public accommodation." § 12182(a).
The term "public accommodation" is defined to include the "professional office of a health care provider." § 12181(7)(F).

A later subsection qualifies the mandate not to discriminate. It provides:

"Nothing in this subchapter shall require an entity to permit an individual to participate in or benefit from the goods, services, facilities, privileges, advantages and accommodations of such entity where such individual poses a direct threat to the health or safety of others." § 12182(b)(3).

The United States and the Maine Human Rights Commission intervened as plaintiffs. After discovery, the parties filed cross-motions for summary judgment. The District Court ruled in favor of the plaintiffs, holding that respondent's HIV infection satisfied the ADA's definition of disability. 912 F.Supp. 580, 585-587 (D.Me.1995). The court held further that petitioner raised no genuine issue of material fact as to whether respondent's HIV infection would have posed a direct threat to

the health or safety of others during the course of a dental treatment. Id., at 587-591. The court relied on affidavits submitted by Dr. Donald Wayne Marianos, Director of the Division of Oral Health of the Centers for Disease Control and Prevention (CDC). The Marianos affidavits asserted it is safe for dentists to treat patients infected with HIV in dental offices if the dentist follows the so- called universal precautions described in the Recommended Infection-Control Practices for Dentistry issued by CDC in 1993 (1993 CDC Dentistry Guidelines). 912 F.Supp., at 589.

The Court of Appeals affirmed. It held respondent's HIV infection was a disability under the ADA, even though her infection had not yet progressed to the symptomatic stage. 107 F.3d 934, 939-943 (C.A.1 1997). The Court of Appeals also agreed that treating the respondent in petitioner's office would not have posed a direct threat to the health and safety of others. Id., at 943-948. Unlike the District Court, however, the Court of Appeals declined to rely on the Marianos affidavits. Id., at 946, n. 7. Instead the court relied on the 1993 CDC Dentistry Guidelines, as well as the Policy on AIDS, HIV Infection and the Practice of Dentistry, promulgated by the American Dental Association in 1991 (1991 American Dental Association Policy on HIV). 107 F.3d, at 945-946.

II

* * *

III

The petition for certiorari presented three other questions for review. The questions stated:

"3. When deciding under title III of the ADA whether a private health care provider must perform invasive procedures on an infectious patient in his office, should courts defer to the health care provider's professional judgment, as long as it is reasonable *2210 in light of then-current medical knowledge?

"4. What is the proper standard of judicial review under title III of the ADA of a private health care provider's judgment that the performance of certain invasive procedures in his office would pose a direct threat to the health or safety of others?

"5. Did petitioner, Randon Bragdon, D. M. D., raise a genuine issue of fact for trial as to whether he was warranted in his judgment that the performance of certain invasive procedures on a patient in his office would have posed a direct threat to the health or safety of others?" Pet. for Cert. i.

Of these, we granted certiorari only on question three. The question is phrased in an awkward way, for it conflates two separate inquiries. In asking whether it is appropriate to defer to petitioner's judgment, it assumes that petitioner's assessment of the objective facts was reasonable. The central premise of the question and the assumption on which it is based merit separate consideration.

Again, we begin with the statute. Notwithstanding the protection given respondent by the ADA's definition of disability, petitioner could have refused to treat her if her infectious condition "pose[d] a direct threat to the health or safety of others." 42 U.S.C. § 12182(b)(3). The ADA defines a direct threat to be "a significant risk to the health or safety of others that cannot be eliminated by a modification of policies, practices, or procedures or by the provision of auxiliary aids or services." Ibid. Parallel provisions appear in the employment provisions of Title I. §§ 12111(3), 12113(b).

The ADA's direct threat provision stems from the recognition in School Bd. of Nassau Cty. v. Arline, 480 U.S. 273, 287, 107 S.Ct. 1123, 1130-1131, 94 L.Ed.2d 307 (1987), of the importance of prohibiting discrimination against individuals with disabilities while protecting others from significant health and safety risks, resulting, for instance, from a contagious disease. In Arline, the Court reconciled these objectives by construing the Rehabilitation Act not to require the hiring of a person who posed "a significant risk of communicating an infectious disease to others." Id., at 287, n. 16, 107 S.Ct., at 1131, n. 16. Congress

amended the Rehabilitation Act and the Fair Housing Act to incorporate the language. See 29 U.S.C. § 706(8)(D) (excluding individuals who "would constitute a direct threat to the health or safety of other individuals"); 42 U.S.C. § 3604(f)(9) (same). It later relied on the same language in enacting the ADA. See 28 CFR pt. 36, App. B, p. 626 (1997) (ADA's direct threat provision codifies Arline). Because few, if any, activities in life are risk free, Arline and the ADA do not ask whether a risk exists, but whether it is significant. Arline, supra, at 287, and n. 16, 107 S.Ct., at 1131, and n. 16; 42 U.S.C. § 12182(b)(3).

[12] The existence, or nonexistence, of a significant risk must be determined from the standpoint of the person who refuses the treatment or accommodation, and the risk assessment must be based on medical or other objective evidence. Arline, supra, at 288, 107 S.Ct., at 1131; 28 CFR § 36.208(c) (1997); id., pt. 36, App. B, p. 626. As a health care professional, petitioner had the duty to assess the risk of infection based on the objective, scientific information available to him and others in his profession. His belief that a significant risk existed, even if maintained in good faith, would not relieve him from liability. To use the words of the question presented, petitioner receives no special deference simply because he is a health care professional. It is true that Arline reserved "the question whether courts should also defer to the reasonable medical judgments of private physicians on which an employer has relied." 480 U.S., at 288, n. 18, 107 S.Ct., at 1131, n. 18. At most, this statement reserved the possibility that employers could consult with individual physicians as objective third-party experts. It did not suggest that an individual physician's state of mind could excuse discrimination without regard to the objective reasonableness of his actions.

Our conclusion that courts should assess the objective reasonableness of the views of health care professionals without deferring to their individual judgments does not answer *2211 the implicit assumption in the question presented, whether petitioner's actions were reasonable in light of the available

medical evidence. In assessing the reasonableness of petitioner's actions, the views of public health authorities, such as the U.S. Public Health Service, CDC, and the National Institutes of Health, are of special weight and authority. Arline, supra, at 288, 107 S.Ct., at 1130-1131; 28 CFR pt. 36, App. B, p. 626 (1997). The views of these organizations are not conclusive, however. A health care professional who disagrees with the prevailing medical consensus may refute it by citing a credible scientific basis for deviating from the accepted norm. See W. Keeton, D. Dobbs, R. Keeton, & D. Owen, Prosser and Keeton on Law of Torts § 32, p. 187 (5th ed.1984).

We have reviewed so much of the record as necessary to illustrate the application of the rule to the facts of this case. For the most part, the Court of Appeals followed the proper standard in evaluating the petitioner's position and conducted a thorough review of the evidence. Its rejection of the District Court's reliance on the Marianos affidavits was a correct application of the principle that petitioner's actions must be evaluated in light of the available, objective evidence. The record did not show that CDC had published the conclusion set out in the affidavits at the time petitioner refused to treat respondent. 107 F.3d, at 946, n. 7.

A further illustration of a correct application of the objective standard is the Court of Appeals' refusal to give weight to the petitioner's offer to treat respondent in a hospital. Id., at 943, n. 4. Petitioner testified that he believed hospitals had safety measures, such as air filtration, ultraviolet lights, and respirators, which would reduce the risk of HIV transmission. App. 151. Petitioner made no showing, however, that any area hospital had these safeguards or even that he had hospital privileges. Id., at 31. His expert also admitted the lack of any scientific basis for the conclusion that these measures would lower the risk of transmission. Id., at 209. Petitioner failed to present any objective, medical evidence showing that treating respondent in a hospital would be safer or more efficient in preventing HIV transmission than treatment in a well-equipped dental office.

We are concerned, however, that the Court of Appeals might have placed mistaken reliance upon two other sources. In ruling no triable issue of fact existed on this point, the Court of Appeals relied on the 1993 CDC Dentistry Guidelines and the 1991 American Dental Association Policy on HIV. 107 F.3d, at 945-946. This evidence is not definitive. As noted earlier, the CDC Guidelines recommended certain universal precautions which, in CDC's view, "should reduce the risk of disease transmission in the dental environment." U.S. Dept. of Health and Human Services, Public Health Service, CDC, Recommended Infection Control Practices for Dentistry, 41 Morbidity & Mortality Weekly Rep. No. RR-18, p. 1 (May 28, 1993). The Court of Appeals determined that, "[w]hile the guidelines do not state explicitly that no further risk- reduction measures are desirable or that routine dental care for HIV-positive individuals is safe, those two conclusions seem to be implicit in the guidelines' detailed delineation of procedures for office treatment of HIV-positive patients." 107 F.3d, at 946. In our view, the Guidelines do not necessarily contain implicit assumptions conclusive of the point to be decided. The Guidelines set out CDC's recommendation that the universal precautions are the best way to combat the risk of HIV transmission. They do not assess the level of risk.

Nor can we be certain, on this record, whether the 1991 American Dental Association Policy on HIV carries the weight the Court of Appeals attributed to it. The Policy does provide some evidence of the medical community's objective assessment of the risks posed by treating people infected with HIV in dental offices. It indicates:

"Current scientific and epidemiologic evidence indicates that there is little risk of transmission of infectious diseases through dental treatment if recommended infection control procedures are routinely followed. Patients with HIV infection may be safely treated in private dental offices when appropriate infection control procedures are employed. Such infection

control procedures *2212 provide protection both for patients and dental personnel." App. 225.

We note, however, that the Association is a professional organization, which, although a respected source of information on the dental profession, is not a public health authority. It is not clear the extent to which the Policy was based on the Association's assessment of dentists' ethical and professional duties in addition to its scientific assessment of the risk to which the ADA refers. Efforts to clarify dentists' ethical obligations and to encourage dentists to treat patients with HIV infection with compassion may be commendable, but the question under the statute is one of statistical likelihood, not professional responsibility. Without more information on the manner in which the American Dental Association formulated this Policy, we are unable to determine the Policy's value in evaluating whether petitioner's assessment of the risks was reasonable as a matter of law.

The court considered materials submitted by both parties on the cross motions for summary judgment. The petitioner was required to establish that there existed a genuine issue of material fact. Evidence which was merely colorable or not significantly probative would not have been sufficient. Anderson v. Liberty Lobby, Inc., 477 U.S. 242, 249-250, 106 S.Ct. 2505, 2510-2511, 91 L.Ed.2d 202 (1986).

We acknowledge the presence of other evidence in the record before the Court of Appeals which, subject to further arguments and examination, might support affirmance of the trial court's ruling. For instance, the record contains substantial testimony from numerous health experts indicating that it is safe to treat patients infected with HIV in dental offices. App. 66-68, 88-90, 264- 266, 268. We are unable to determine the import of this evidence, however. The record does not disclose whether the expert testimony submitted by respondent turned on evidence available in September 1994. See id., at 69- 70 (expert

testimony relied in part on materials published after September 1994).

There are reasons to doubt whether petitioner advanced evidence sufficient to raise a triable issue of fact on the significance of the risk. Petitioner relied on two principal points: First, he asserted that the use of high-speed drills and surface cooling with water created a risk of airborne HIV transmission. The study on which petitioner relied was inconclusive, however, determining only that "[f]urther work is required to determine whether such a risk exists." Johnson & Robinson, Human Immunodeficiency Virus-1 (HIV-1) in the Vapors of Surgical Power Instruments, 33 J. of Medical Virology 47, 47 (1991). Petitioner's expert witness conceded, moreover, that no evidence suggested the spray could transmit HIV. His opinion on airborne risk was based on the absence of contrary evidence, not on positive data. App. 166. Scientific evidence and expert testimony must have a traceable, analytical basis in objective fact before it may be considered on summary judgment. See General Electric Co. v. Joiner, 522 U.S. 136, ----, ----, 118 S.Ct. 512, 518, 519, 139 L.Ed.2d 508 (1997).

Second, petitioner argues that, as of September 1994, CDC had identified seven dental workers with possible occupational transmission of HIV. See U.S. Dept. of Health and Human Services, Public Health Service, CDC, HIV/AIDS Surveillance Report, vol. 6, no. 1, p. 15, tbl. 11 (Mid-year ed. June 1994). These dental workers were exposed to HIV in the course of their employment, but CDC could not determine whether HIV infection had resulted. Id., at 15, n. 3. It is now known that CDC could not ascertain whether the seven dental workers contracted the disease because they did not present themselves for HIV testing at an appropriate time after their initial exposure. Gooch et al., Percutaneous Exposures to HIV-Infected Blood Among Dental Workers Enrolled in the CDC Needlestick Study, 126 J. American Dental Assn. 1237, 1239 (1995). It is not clear on this record, however, whether this information was available to petitioner in September 1994. If not, the seven cases might

have provided some, albeit not necessarily sufficient, support for petitioner's position. Standing alone, we doubt it would meet the objective, scientific basis for finding a significant risk to the petitioner.

*2213 Our evaluation of the evidence is constrained by the fact that on these and other points we have not had briefs and arguments directed to the entire record. In accepting the case for review, we declined to grant certiorari on question five, which asked whether petitioner raised a genuine issue of fact for trial. Pet. for Cert. i. As a result, the briefs and arguments presented to us did not concentrate on the question of sufficiency in light all of the submissions in the summary judgment proceeding. "When attention has been focused on other issues, or when the court from which a case comes has expressed no views on a controlling question, it may be appropriate to remand the case rather than deal with the merits of that question in this Court." Dandridge v. Williams, 397 U.S. 471, 476, n. 6, 90 S.Ct. 1153, 1157, n. 6, 25 L.Ed.2d 491 (1970). This consideration carries particular force where, as here, full briefing directed at the issue would help place a complex factual record in proper perspective. Resolution of the issue will be of importance to health care workers not just for the result but also for the precision and comprehensiveness of the reasons given for the decision.

We conclude the proper course is to give the Court of Appeals the opportunity to determine whether our analysis of some of the studies cited by the parties would change its conclusion that petitioner presented neither objective evidence nor a triable issue of fact on the question of risk. In remanding the case, we do not foreclose the possibility that the Court of Appeals may reach the same conclusion it did earlier. A remand will permit a full exploration of the issue through the adversary process.

The determination of the Court of Appeals that respondent's HIV infection was a disability under the ADA is affirmed. The judgment is vacated, and the case is remanded for further proceedings consistent with this opinion.

It is so ordered.

Justice STEVENS, with whom Justice BREYER joins, concurring.

The Court's opinion demonstrates that respondent's HIV infection easily falls within the statute's definition of "disability." Moreover, the Court's discussion in Part III of the relevant evidence has persuaded me that the judgment of the Court of Appeals should be affirmed. I do not believe petitioner has sustained his burden of adducing evidence sufficient to raise a triable issue of fact on the significance of the risk posed by treating respondent in his office. The Court of Appeals reached that conclusion after a careful and extensive study of the record; its analysis on this question was perfectly consistent with the legal reasoning in Justice KENNEDY's opinion for the Court; and the latter opinion itself explains that petitioner relied on data that was inconclusive and speculative at best, see ante, at 2212-2213. Cf. General Electric Co. v. Joiner, 522 U.S. 136, 118 S.Ct. 512, 139 L.Ed.2d 508 (1997).

There are not, however, five Justices who agree that the judgment should be affirmed. Nor does it appear that there are five Justices who favor a remand for further proceedings consistent with the views expressed in either Justice KENNEDY's opinion for the Court or the opinion of THE CHIEF JUSTICE. Because I am in agreement with the legal analysis in Justice KENNEDY's opinion, in order to provide a judgment supported by a majority, I join that opinion even though I would prefer an outright affirmance. Cf. Screws v. United States, 325 U.S. 91, 134, 65 S.Ct. 1031, 1051, 89 L.Ed. 1495 (1945) (Rutledge, J., concurring in result).

Justice GINSBURG, concurring.

HIV infection, as the description set out in the Court's opinion documents, ante, at 2203-2205, has been regarded as a disease

limiting life itself. See Brief for American Medical Association as Amicus Curiae 20. The disease inevitably pervades life's choices: education, employment, family and financial undertakings. It affects the need for and, as this case shows, the ability to obtain health care because of the reaction of others to the impairment. No rational legislator, it seems to me apparent, would require nondiscrimination once symptoms become visible but permit discrimination when the disease, ***2214** though present, is not yet visible. I am therefore satisfied that the statutory and regulatory definitions are well met. HIV infection is "a physical ... impairment that substantially limits ... major life activities," or is so perceived, 42 U.S.C. §§ 12102(2)(A),(C), including the afflicted individual's family relations, employment potential, and ability to care for herself, see 45 CFR § 84.3(j)(2)(ii) (1997); 28 CFR § 41.31(b)(2) (1997).

I further agree, in view of the "importance [of the issue] to health care workers," ante, at 2213, that it is wise to remand, erring, if at all, on the side of caution. By taking this course, the Court ensures a fully informed determination whether respondent Abbott's disease posed "a significant risk to the health or safety of [petitioner Bragdon] that [could not] be eliminated by a modification of policies, practices, or procedures...." 42 U.S.C. § 12182(b)(3).

Chief Justice REHNQUIST, with whom Justice SCALIA and Justice THOMAS join, and with whom Justice O'CONNOR joins as to Part II, concurring in the judgment in part and dissenting in part.

I

* * *

II

While the Court concludes to the contrary as to the "disability" issue, it then quite correctly recognizes that petitioner could nonetheless have refused to treat respondent if her condition

posed a "direct threat." The Court of Appeals affirmed the judgment of the District Court granting summary judgment to respondent on this issue. The Court vacates this portion of the Court of Appeals' decision, and remands the case to the lower court, presumably so that it may "determine whether our analysis of some of the studies cited by the parties would change its conclusion that petitioner presented neither objective evidence nor a triable issue of fact on the question of risk." Ante, at 2213. I agree that the judgment should be vacated, although I am not sure I understand the Court's cryptic direction to the lower court.

"[D]irect threat" is defined as a "significant risk to the health or safety of others that cannot be eliminated by a modification of policies, practices, or procedures or by the provision of auxiliary aides or services." § 12182(b)(3). This statutory definition of a direct threat consists of two parts. First, a court must ask whether treating the infected patient without precautionary techniques would pose a "significant risk to the heath or safety of others." Ibid. Whether a particular risk is significant depends on:

" '(a) the nature of the risk (how the disease is transmitted), (b) the duration of the risk (how long is the carrier infectious), (c) the severity of the risk (what is the potential harm to third parties) and (d) the probabilities the disease will be transmitted and will cause varying degrees of harm.' " School Bd. of Nassau Cty. v. Arline, 480 U.S. 273, 288, 107 S.Ct. 1123, 1131, 94 L.Ed.2d 307 (1987).

Even if a significant risk exists, a health practitioner will still be required to treat the infected patient if "a modification of policies, practices, or procedures" (in this case, universal precautions) will "eliminat[e]" the risk. § 12182(b)(3).

I agree with the Court that "the existence, or nonexistence, of a significant risk must be determined from the standpoint of the person who refuses the treatment or accommodation," as of the time that the decision refusing treatment is made. Ante, at 2210. I disagree with the Court, however, that "[i]n assessing the

reasonableness of petitioner's actions, the views of public health authorities ... are of special weight and authority." Ante, at 2211. Those views are, of course, *2217 entitled to a presumption of validity when the actions of those authorities themselves are challenged in court, and even in disputes between private parties where Congress has committed that dispute to adjudication by a public health authority. But in litigation between private parties originating in the federal courts, I am aware of no provision of law or judicial practice that would require or permit courts to give some scientific views more credence than others simply because they have been endorsed by a politically appointed public health authority (such as the Surgeon General). In litigation of this latter sort, which is what we face here, the credentials of the scientists employed by the public health authority, and the soundness of their studies, must stand on their own. The Court cites no authority for its limitation upon the courts' truth-finding function, except the statement in School Bd. of Nassau Cty. v. Arline, 480 U.S., at 288, 107 S.Ct., at 1131, that in making findings regarding the risk of contagion under the Rehabilitation Act, "courts normally should defer to the reasonable medical judgments of public health officials." But there is appended to that dictum the following footnote, which makes it very clear that the Court was urging respect for medical judgment, and not necessarily respect for "official" medical judgment over "private" medical judgment: "This case does not present, and we do not address, the question whether courts should also defer to the reasonable medical judgments of private physicians on which an employer has relied." Id., at 288, n. 18, 107 S.Ct., at 1131, n. 18.

Applying these principles here, it is clear to me that petitioner has presented more than enough evidence to avoid summary judgment on the "direct threat" question. In June 1994, the Centers for Disease Control and Prevention published a study identifying seven instances of possible transmission of HIV from patients to dental workers. See Ante, at 2212. While it is not entirely certain whether these dental workers contracted HIV during the course of providing dental treatment, the potential that

the disease was transmitted during the course of dental treatment is relevant evidence. One need only demonstrate "risk," not certainty of infection. See Arline, supra, at 288, 107 S.Ct., at 1131 (" 'the probabilities the disease will be transmitted' " is a factor in assessing risk). Given the "severity of the risk" involved here, i.e., near certain death, and the fact that no public health authority had outlined a protocol for eliminating this risk in the context of routine dental treatment, it seems likely that petitioner can establish that it was objectively reasonable for him to conclude that treating respondent in his office posed a "direct threat" to his safety.

In addition, petitioner offered evidence of 42 documented incidents of occupational transmission of HIV to healthcare workers other than dental professionals. App. 106. The Court of Appeals dismissed this evidence as irrelevant because these health professionals were not dentists. 107 F.3d 934, 947 (C.A.1 1997). But the fact that the health care workers were not dentists is no more valid a basis for distinguishing these transmissions of HIV than the fact that the health care workers did not practice in Maine. At a minimum, petitioner's evidence was sufficient to create a triable issue on this question, and summary judgment was accordingly not appropriate.

Justice O'CONNOR, concurring in the judgment in part and dissenting in part.

I agree with THE CHIEF JUSTICE that respondent's claim of disability should be evaluated on an individualized basis and that she has not proven that her asymptomatic HIV status substantially limited one or more of her major life activities. In my view, the act of giving birth to a child, while a very important part of the lives of many women, is not generally the same as the representative major life activities of all persons--"caring for one's self, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working"--listed in regulations relevant to the Americans with Disabilities Act of 1990. See 45 CFR § 84.3(j)(2)(ii) (1997); 28 CFR § 41.31(b)(2)

(1997). Based on that conclusion, there is no need to address whether other aspects of intimate or family relationships not raised in this case could constitute major life activities; nor is there reason to consider whether HIV status ***2218** would impose a substantial limitation on one's ability to reproduce if reproduction were a major life activity.

I join in Part II of THE CHIEF JUSTICE's opinion concurring in the judgment in part and dissenting in part, which concludes that the Court of Appeals failed to properly determine whether respondent's condition posed a direct threat. Accordingly, I agree that a remand is necessary on that issue.

Sidney ABBOTT, et al., Plaintiffs, Appellees,
v.
Randon BRAGDON, D.M.D., Defendant, Appellant.

No. 96-1643.

United States Court of Appeals,
First Circuit.

Heard Nov. 8, 1996.

Decided March 5, 1997.

HIV-positive patient brought action under Americans with Disabilities Act (ADA) and Maine Human Rights Act against dentist who refused to treat her in his office. The United States District Court for the District of Maine, Morton A. Brody, J., 912 F.Supp. 580, granted summary judgment, in favor of patient, and dentist appealed. The Court of Appeals, Selya, Circuit Judge, held, in a case of first impression, that: (1) HIV-positive status comprises "physical impairment" under ADA; (2) reproduction constitutes "major life activity" under ADA; (3) reproduction was "major life activity" under ADA for patient; (4) HIV-positive status is physical impairment that "substantially limits" fecund woman's major life activity of reproduction; (5) neither service provider nor prospective recipient of service may prove or disprove direct threat defense to ADA claim by relying on medical evidence not available when treatment was refused; (6) service provider cannot successfully contradict achieved consensus that particular disability is not a direct threat simply by proffering unsupported opinion; and (7) in September of 1994, reasonable medical judgments by public health officials, was that affording routine dental care to HIV-infected patients in office environment did not pose "direct threat" to dentist's health.

Affirmed.

[West Headnotes Omitted]

*937 John W. McCarthy, Bangor, ME, with whom Brent A. Singer and Rudman & Winchell were on brief, for defendant.

Peter M. Sfikas, Mark S. Rubin and Jill A. Wolowitz, Chicago, IL, on brief for American Dental Ass'n, amicus curiae.

Bennett H. Klein and David G. Webbert, Augusta, ME, with whom Gay and Lesbian Advocates and Johnson, Webbert & Laubenstein were on brief, for plaintiff Sidney Abbott.

John E. Carnes, Commission Counsel, Augusta, ME, on brief for intervenor- plaintiff Maine Human Rights Commission.

Samuel R. Bagenstos, Attorney, U.S. Dept. of Justice, Washington, DC, with whom Deval L. Patrick, Assistant Attorney General, Boston, MA, and Jessica Dunsay Silver, Attorney, Washington, DC, were on brief, for intervenor-plaintiff United States of America, amicus curiae.

Robert Greenwald, Yonkers, NY, and AIDS Action Committee on brief, Boston, MA, for R.I. Dept. of Pub. Health, Bureau of Health of the Me. Dept. of Human Servs., American Pub. Health Ass'n, Ass'n of State and Territorial Dental Dirs., and Ass'n of State and Territorial Health Officials, amici curiae.

Donna Levin, Boston, MA, on brief for Mass. Dept. of Pub. Health, amicus curiae.

Before SELYA, Circuit Judge, ALDRICH, Senior Circuit Judge, and STAHL, Circuit Judge.

SELYA, Circuit Judge.

In this case of first impression, the district court granted summary judgment in favor of plaintiff-appellee Sidney Abbott, an HIV-positive woman, on her-claim that defendant-appellant Randon Bragdon, a dentist, violated the Americans with Disabilities Act (the ADA) by refusing to treat her in his dental office. [FN1] Dr. Bragdon appeals. Because we agree with the district court that Ms. Abbott is disabled within the purview of the ADA and that providing routine dental care to her (i.e., filling a cavity) would not have posed a direct threat to Dr. Bragdon's health or safety, we affirm.

FN1. Though we write for simplicity's sake as if Ms. Abbott were the sole plaintiff, we note that the federal government and the Maine Human Rights Commission intervened as plaintiffs below. We note, too, that Ms. Abbott prevailed upon a parallel claim under the Maine Human Rights Act (MHRA), 5 Me.Rev.Stat.Ann. tit. 5, § 4592 (West 1989). Interpretation of both the ADA and the MHRA has "proceeded hand in hand," Soileau v. Guilford of Me., Inc., 105 F.3d 12, 14 (1st Cir.1997), and the parties here do not suggest any distinction between the two statutes that might affect this appeal. Consequently, we need not discuss the MHRA further.

I. BACKGROUND

The events giving rise to this litigation are straightforward. On September 16, 1994, Ms. Abbott arrived at Dr. Bragdon's office in Bangor, Maine for a scheduled appointment. On her patient registration form, she indicated that she was infected with the HIV virus. People may be HIV-positive for years

without manifesting the set of symptoms commonly known as AIDS, and Ms. Abbott was asymptomatic at the time.

Dr. Bragdon performed a dental examination and discovered a cavity. He told Ms. Abbott that, pursuant to his infectious disease policy, he would not fill her cavity in his office, but would only treat her in a hospital setting. Though he would charge his regular fee, she would have to bear the additional cost of whatever the hospital charged for the use of its facilities. Ms. Abbott refused the ***938** offer and instead filed a complaint under the ADA. See 42 U.S.C. § 12182(a) (1994).

After pretrial discovery concluded, the parties cross-moved for summary judgment. The district court ruled that Ms. Abbott was substantially limited in a major life activity (reproduction) and thus was disabled for purposes of the ADA. See Abbott v. Bragdon, 912 F.Supp. 580, 587 (D.Me.1995). The court then concluded that the relatively routine treatment needed by Ms. Abbott could be delivered safely in Dr. Bragdon's office. See id. at 591. Consequently, the court granted Ms. Abbott's motion for summary judgment. See id. at 595-96. This appeal followed.

II. THE SUMMARY JUDGMENT STANDARD

[1] The Civil Rules authorize federal courts to grant summary judgment only when "there is no genuine issue as to any material fact and ... the moving party is entitled to judgment as a matter of law." Fed.R.Civ.P. 56(c). Confronted with a properly documented motion for brevis disposition, the nonmovant must establish the existence of a fact that is both genuine and material in order to ward off the entry of an adverse judgment. See Anderson v. Liberty Lobby, Inc., 477 U.S. 242, 248, 106 S.Ct. 2505, 2510, 91 L.Ed.2d 202 (1986); Garside v. Osco Drug, Inc., 895 F.2d 46, 48 (1st Cir.1990). Appellate review of the district court's award of summary

judgment is plenary, and, in keeping with this standard, we are not wedded to the district court's rationale but may affirm on any alternative ground made manifest by the record. See Hachikian v. FDIC, 96 F.3d 502, 504 (1st Cir.1996).

[2] In assaying the record, we are guided by the same tenets that guided the lower court. Thus, we are duty bound to indulge all reasonable inferences in favor of the party opposing summary judgment. See id. This generous outlook notwithstanding, we must disregard improbable or overly attenuated inferences, unsupported conclusions, and rank speculation. See Smith v. F.W. Morse & Co., 76 F.3d 413, 428 (1st Cir.1996); Medina-Munoz v. R.J. Reynolds Tobacco Co., 896 F.2d 5, 8 (1st Cir.1990).

III. THE QUESTION OF DISABILITY

* * *

IV. THE DIRECT THREAT QUESTION

Under the ADA, a place of public accommodation must extend its services to all disabled persons without reference to their disabilities, subject to certain carefully circumscribed exceptions. See 42 U.S.C. § 12182(a). By virtue of one such exception, a covered service provider need not deal with an individual who "poses a direct threat to the health or safety of others." 42 U.S.C. § 12182(b)(3). The term "direct threat" is defined by the statute; in this context it contemplates the existence of "a significant risk to the health or safety of others that cannot be eliminated by a modification of policies, practices, or procedures or by the provision of auxiliary aids or services." Id. [FN4]

FN4. It is important to note that Dr. Bragdon does not argue that his offer to treat Ms. Abbott in a hospital setting is a

practice modification that would eliminate the claimed threat to his health. He did not endeavor to prove in the court below that hospital treatment was significantly safer than office treatment, and he makes no such argument to us. He argues instead that he had no duty to treat her in his office and that his offer to treat her in a hospital was gratuitous (and, therefore, legally irrelevant). Dr. Bragdon claims the right to deny entirely routine dental treatment to patients with HIV, and his defense must stand or fall on the correctness vel non of that thesis. It is only that claim which we consider here. Thus, this case does not address whether it is illegal under the ADA for dentists to take additional precautions when treating HIV-infected patients.

Dr. Bragdon seeks safe harbor under this exception, asserting that requiring him to treat Ms. Abbott would pose a direct threat to his health. His argument envisions that to fill Ms. Abbott's cavity, he would need to inject a local anesthetic into her mouth and drill the decayed tooth. These procedures, he says, create an undue risk of HIV transmission both through needlestick and through the spattering of blood and bloody saliva.

A court's goal in conducting a direct threat analysis under the ADA is to achieve a responsible balance, protecting service providers and other places of accommodation from enforced exposure to unacceptable health and safety risks while at the same time protecting disabled individuals from discrimination that is rooted in prejudice or baseless fear. See Arline, 480 U.S. at 287, 107 S.Ct. at 1130-31. EEOC regulations drawn from the Court's seminal opinion in Arline guide this analysis:

> In determining whether an individual poses a direct threat to the health or safety of others, a public accommodation must make an individualized assessment, based on reasonable judgment that relies on current medical knowledge or on the best available objective evidence, to

ascertain: the nature, duration, and severity of the risk; the probability that the potential injury will actually occur; and whether reasonable modifications of policies, practices, or procedures will mitigate the risk.

28 C.F.R. § 36.208(c) (1996); see also Arline, 480 U.S. at 287-88, 107 S.Ct. at 1130-31 (discussing essentially the same factors).

Of course, any inquiry along these lines depends in one sense on what information permissibly may be weighed in the balance and in another sense on the extent to which particular kinds of evidence should be assigned extra weight (or, perhaps, decretory significance). Thus, before evaluating the medical evidence proffered by the parties, we must determine (1) the relevancy, if any, of subsequent medical knowledge (that is, medical evidence not available in September 1994 when Dr. Bragdon refused to treat Ms. Abbott), and (2) the degree of deference, if any, due the judgments of public health officials.

*944 A. The Evidentiary Time Line.

[16] The first of these excursions need not detain us. The applicable regulations instruct that a judgment on the presence or absence of a direct threat must be predicated on "current medical knowledge or on the best available objective evidence," 28 C.F.R. § 36.208(c) (1996) (emphasis supplied), and, hence, point unwaveringly toward confining medical evidence to that available at the time a dentist or other health-care professional refuses to treat. This principle--that neither the service provider nor the prospective recipient of the service may prove or disprove the direct threat defense by relying on medical evidence not available when treatment was refused--is sound policy. Under such a regime, the service provider retains the opportunity to prove that he made an appropriate determination of the existence of a direct threat based on the

evidence available when he made the decision to withhold his services. This is sound policy because, while health- care providers can be expected to maintain a working knowledge of currently accepted thinking in their fields, they cannot be expected to anticipate either future scientific advances or the emerging wisdom of public health organizations. In short, by holding covered service providers to an objective standard featuring the best evidence available at the time they refuse to render treatment to disabled persons, the ADA holds the delicate balance between sometimes conflicting rights steady and true.

This approach is also scrupulously fair. To punish providers when they satisfy an objective standard based on the best evidence available at the time of their decisions would be to punish them for a lack of clairvoyance. By the same token, to hold providers harmless after they have refused treatment based on nothing more than unfounded trepidation would run at cross-purposes with the central theme of the ADA. Fundamental fairness insists that providers in such circumstances ought not to be entitled to rely on subsequent understandings to shield them from the condign consequences of discriminatory conduct. [FN5] Cf. McKennon v. Nashville Banner Pub. Co., 513 U.S. 352, ----, 115 S.Ct. 879, 885, 130 L.Ed.2d 852 (1995) (holding that an employer in an employment discrimination case may not justify its conduct based on evidence that did not motivate it at the time of the employment decision); North Shore Univ. Hosp. v. Rosa, 86 N.Y.2d 413, 633 N.Y.S.2d 462, 465, 657 N.E.2d 483, 486 (1995) (evaluating conduct alleged to be discriminatory under state law based on accepted medical practice at the time of the alleged infraction).

FN5. Few rules are without exception in extreme cases. One can conceive of situations in which, after a medical professional refused treatment but before trial, there might

occur some medical breakthrough or stunning new CDC pronouncement that overwhelmingly vindicated the professional's fears. Especially because decisions in ADA cases have some forward-looking impact, we do not rule out the possibility that later occurring events might occasionally be pertinent. But nothing in the present record suggests any post hoc scientific revelation warranting a departure from the usual rule.

B. The Degree of Deference.

[17] The second threshold determination involves the degree of deference due the medical judgments of public health authorities. The government joins Ms. Abbott in arguing for a rule which, if embraced, would cede great deference to those authorities. They posit that, in the absence of dissent among public health authorities, a service provider should be bound to accept the expressed collective judgment of those authorities unless he can demonstrate that this judgment is medically unreasonable. In contrast, Dr. Bragdon asseverates that, at least in the case of a service provider who is himself a skilled professional (such as a doctor or dentist), a court should defer to the provider's judgment, as long as it appears to have been reasonable in light of then-current medical knowledge.

The question of deference pivots on language in Arline, where Justice O'Connor, writing for the Court, stated that in making factual findings of the sort that are necessary to inform the inquiry into the existence vel non of a direct threat, "courts normally should defer to the reasonable medical judgments *945 of public health officials." 480 U.S. at 288, 107 S.Ct. at 1131. In crafting regulations applicable to the ADA, the EEOC treated this passage from Arline as gospel. See 28 C.F.R. Pt. 36, App. B § 36.208 (1996) (remarking that the direct threat regulations "codif[y] the standard first applied by the Supreme Court in [Arline]").

We agree that the deference due public health officials must flow from the quoted passage--but acknowledging the hegemony of Arline does not signal automatic victory for Ms. Abbott and the government. The "defer entirely" formulation that they urge upon us is totally unprecedented; we have found no case in which the views of public health authorities are treated with the solicitude that Ms. Abbott and the government invite. If adopted, this formulation would come close to making a consensus among public health authorities unchallengeable by other medical evidence and, consequently, unreviewable by the courts. Nothing in Arline demands such obsequious obeisance to public health authorities or indicates an intention on the Court's part to consign the medical judgments of private physicians to some evidentiary Siberia. Instead, the Court quite clearly left the details of deference for another day. See, e.g., Arline, 480 U.S. at 288 n. 18, 107 S.Ct. at 1131 n. 18 ("This case does not present, and we do not address, the question whether courts should also defer to the reasonable medical judgments of private physicians....").

Because we are unprepared to say that medical wisdom resides exclusively in public health authorities, we reject the idea of a conclusive presumption. The applicable regulations state that "[s]ources for medical knowledge include guidance from public health authorities, such as the U.S. Public Health Service, the Centers for Disease Control, and the National Institutes of Health." 28 C.F.R. Pt. 36, App. B § 36.208 (1996) (emphasis supplied). This list is plainly illustrative, not exhaustive, and the use of the verb "include" indicates to us that other sources of medical knowledge are within the pale. The statute, the suggestion implicit in the regulations, and the teachings of the Court are best synthesized by fashioning a rule which gives prima facie force to the views of public health authorities, but which permits a service provider to challenge those views based on contrary, properly supported

opinions voiced by other recognized experts in the field (e.g., research studies published in peer-reviewed journals). Such a rule accords a meaningful degree of respect to the views of public health authorities, particularly when those views are unanimous. But the rule draws a distinction between respect and absolute capitulation. Under it, the conclusions of public health authorities may be rebutted by persuasive evidence adduced from other recognized experts in a given field. [FN6]

FN6. In United States v. Jessup, 757 F.2d 378, 381-84 (1st Cir.1985), then-Judge Breyer distinguished "bursting bubble" presumptions (which vanish when contrary evidence is introduced) from "intermediate" presumptions (which remain available for consideration by the factfinder even after contrary evidence is introduced). The presumption here is of the latter stripe.

[18] Treating the presumption of correctness which attaches to the collective judgment of public health authorities as rebuttable will not, as the government intimates, sabotage the statutory scheme. Because the test for the existence vel non of a direct threat remains an objective one, a service provider cannot successfully contradict an achieved consensus simply by proffering an unsupported opinion. This ensures that, despite the rebuttable nature of the presumption, the direct threat defense may not be used to mask prejudice or unfounded fears. Rather, to frame a genuine issue, an opposing view must be documented by competent countervailing evidence that is directly relevant. Speculative inferences, glancing statistics, unsupported conclusions, and ruminative surmise will not serve.

C. Applying the Standard.

[19] We turn next to a review of the medical evidence that was available when Ms. Abbott visited Dr. Bragdon's office in

September 1994. By then, both the United States Centers for Disease Control and Prevention (CDC) and the American Dental Association (the Association) had spoken to the issue of the health risk to dental workers from patients infected with HIV. The Association's 1991 Policy on AIDS, HIV Infection and the Practice of Dentistry stated that:

***946** Current scientific and epidemiologic evidence indicates that there is little risk of transmission of infectious diseases through dental treatment if recommended infection control procedures are routinely followed. Patients with HIV infection may be safely treated in private dental offices when appropriate infection control procedures are employed.

In 1993, the CDC updated its earlier guidelines and specified a compendium of infection control procedures, known as the "universal precautions," for use by dental workers treating HIV-positive patients. See CDC, Recommended Infection-Control Practices for Dentistry, 1993 (the Recommendations). The CDC took the position that, when implemented, the prescribed precautions "should reduce the risk of disease transmission in the dental environment." Id. at 3. While the guidelines do not state explicitly that no further risk-reduction measures are desirable or that routine dental care for HIV-positive individuals is safe, those two conclusions seem to be implicit in the guidelines' detailed delineation of procedures for office treatment of HIV-positive patients. See United States v. Morvant, 898 F.Supp. 1157, 1166 (E.D.La.1995) (concluding that "the universal precautions as prescribed by the CDC are universally accepted as 'reasonable modifications' of practices that will significantly mitigate the risk [of HIV transmission from patient to dentist]"). [FN7] Tellingly, no public health authority has suggested that it is unsafe to provide routine dental care to HIV-positive patients in a private office environment. We find, therefore, that Ms. Abbott adduced competent evidence of reasonable medical judgments by public health officials, not contradicted by other public health authorities, to the effect that affording routine

dental care (such as filling cavities) to HIV-infected patients in an office environment does not pose a direct threat to the dentist's health.

FN7. In support of her motion for brevis disposition, Ms. Abbott also presented testimony from Dr. Donald Wayne Marianos, director of the Division of Oral Health at the CDC. Dr. Marianos stated categorically that "[n]o infection control procedures beyond the use of universal precautions are necessary when providing routine dental care to persons with HIV and AIDS." He also declared that "[t]he CDC does not recommend the use of infection control procedures beyond those cited in [the Recommendations] for the provision of routine dental treatment to persons with HIV or AIDS" and that "the risk of HIV transmission from patient to provider [in such circumstances] is so low as to be unquantifiable." Dr. Marianos' testimony is of limited value to us in assessing the medical evidence available to Dr. Bragdon inasmuch as the record contains no evidence that the CDC had publicly taken so explicit a position as of September 1994.

The next question is whether Dr. Bragdon has produced sufficient countervailing evidence that filling Ms. Abbott's cavity in an office setting would have constituted a direct threat to his health. In an endeavor to create a genuine issue of material fact, he cites eight sources of information which he argues show that rendering the necessary treatment in his office would have jeopardized his health. [FN8] We examine these sources to determine if any of them, individually or in the aggregate, justify denying summary judgment to Ms. Abbott.

FN8. Although Dr. Bragdon presented material from a retained expert, Dr. Sanford Kuvin, he does not claim that Dr. Kuvin's testimony was based on medical knowledge available to him on the date he declined to treat Ms. Abbott, and his

brief discusses Dr. Kuvin's testimony in a separate section devoted to the possibility that evidence available after September of 1994 might be relevant to the issue. To clinch the point, Dr. Kuvin's testimony itself relies on a number of sources not available until 1995 and beyond.

1. Dr. Bragdon notes that the Food and Drug Administration (the FDA) recommended in 1992 that persons who have had contact with a patient's blood through needlestick, non-intact skin, or mucous membranes refrain from donating blood for a year. This recommendation is clearly insufficient to demonstrate a direct threat to Dr. Bragdon. To safeguard the integrity of the blood supply, the FDA may seek to avoid minute risks and take unusual precautions even in the absence of actual evidence of danger. It is not surprising, therefore, that the FDA issued its recommendation without making any finding that there was a significant risk of contracting HIV from contact of the type and kind described. In contrast, to determine whether there is a direct threat within the purview of the ADA, an inquiring court must gauge the nature, duration, and ***947** severity of the risk. [FN9] The FDA's recommendation does not advance this inquiry.

FN9. Then, too, the stakes are different. The only foreseeable loss from what may be an overabundance of caution on the FDA's part is some small quantity of donated blood. Surrendering to stereotypes and unfounded fears when dealing with disabled persons, however, will subvert Congress' intent in enacting the ADA.

2. Dr. Bragdon refers to a CDC report documenting forty-two incidents of transmission of HIV to health-care workers and seven possible transmissions to dental workers. We deem this data insufficient to warrant depriving Ms. Abbott of summary judgment. Evidence of HIV transmission to health-care workers outside the dental field does not prove a direct threat

to a practicing dentist in the absence of any evidence showing that the magnitude of risk to a dentist is comparable to the risk to other health-care workers in other settings. Nor is such an equivalency obvious; health-care workers in, say, emergency rooms may be exposed to much larger needles and more copious quantities of blood than are common in routine dental practice.

Generalities about health-care workers aside, Dr. Bragdon does not cite a single confirmed instance of HIV transmission to a dentist. He does, of course, point to seven instances of "possible transmissions" of HIV to dental workers, but mere possibilities are too speculative to satisfy a litigant's burden of production at the summary judgment stage. See Smith, 76 F.3d at 428 (noting that unsupported speculation must be disregarded at summary judgment); Medina-Munoz, 896 F.2d at 8 (similar).

3. Dr. Bragdon brandishes a CDC report telling of the transmission of HIV from a Florida dentist to his patients. But neither the CDC report nor any other proffered evidence establishes transmission of HIV from a patient to a dental worker. The difference is meaningful. Dentists have the advantage of equipping and staffing their offices and dictating the precautionary procedures that will be utilized during patient encounters. In contrast, patients have no way of assuring that any particular risk-reduction measures, much less the universal precautions recommended by the CDC, are introduced. Given that dentists are in a superior position to patients in terms of protecting against infection, a single report of dentist-to-patient infection cannot reasonably be taken to suggest a direct threat to dentists from their patients' infections.

4. The high-speed drills that dentists use when filling cavities may also generate aerosol mists of water, blood, and bloody

saliva. Using this datum as a springboard, Dr. Bragdon jumps to the conclusion that a study by researchers at the Stanford Medical School, raising concerns about transmission of HIV when dentists perform aerosol generating procedures, is sufficient to defeat summary judgment. See Gregory K. Johnson and William S. Robinson, Human Immunodeficiency Virus-1 (HIV-1) in the Vapors of Surgical Power Instruments, 33 Journal of Medical Virology 47 (1991). By its own characterization, however, the Johnson-Robinson paper is too conjectural to raise a genuine issue of material fact as to whether filling Ms. Abbott's cavity would constitute a direct threat to Dr. Bragdon's health. The paper notes that it "do[es] not quantitate the risk of HIV transmission ... by such aerosols," and that "the large body of epidemiologic data on prevalence of HIV infections ... would suggest that transmission by aerosols is not common." Id. at 49.

5. Dr. Bragdon points out that the CDC did not state that it was medically unwise to take additional precautions with persons known to be HIV-positive. This observation, while true, gains him little ground. Such silence on the part of the CDC is at best equivocal; it does nothing to prove that there is any risk to a dentist in treating HIV-positive patients. Because the inference that Dr. Bragdon seeks to draw from the CDC's silence is wholly conjectural, it cannot figure in the summary judgment calculus. [FN10] See Smith, 76 F.3d at 428.

FN10. In all events, it is implicit in the 1993 CDC guidelines that no risk-reduction steps beyond the universal precautions are necessary to ensure the safety of dentists providing routine dental care to HIV-positive individuals. See supra p. 945-46.

6. Dr. Bragdon adverts to the Association's report that the risk to health- care workers is greater than the risk to patients. At the same time, he notes, federal courts have found a significant

risk of HIV transmission *948 from health-care workers to patients. See, e.g., Bradley v. University of Tex. M.D. Anderson Cancer Ctr., 3 F.3d 922, 924 (5th Cir.1993) (per curiam), cert. denied, 510 U.S. 1119, 114 S.Ct. 1071, 127 L.Ed.2d 389 (1994); Doe v. Washington Univ., 780 F.Supp. 628, 633 (E.D.Mo.1991). Dr. Bragdon seeks to lace these findings together to support a conclusion that the risk to him from treating Ms. Abbott is also significant. The tie does not bind; this combination produces far too much of a generalization to have any force in the much narrower contours of this case.

Moreover, the federal cases involving transmission of HIV from health-care workers to patients are inapposite not only because of the asymmetry of control of risk-reduction measures between health-care workers and patients, see supra, but also because the cited cases spring from a context in which any risk at all to patients is deemed unacceptable. See Washington Univ., 780 F.Supp. at 633 ("It is the stated goal of the medical profession to heal, and its secondary axiom, if healing is not possible, is not to harm.") Therefore, these cases do not support a reasoned inference that rendering routine care to a dental patient infected with HIV constitutes a direct threat to the dentist's health.

7. Dr. Bragdon states that although he did his best to comply with the universal precautions, he still sustained sharp injuries on a regular basis. We believe that this kind of anecdotal evidence by a dentist who is not an expert on infectious disease is inadequate to block summary judgment. See Medina-Munoz, 896 F.2d at 8 (noting that summary judgment may be granted when opposing evidence is not significantly probative). Although courts need not defer slavishly to the judgments of public health officials, see supra Part IV(B), we believe it would be inconsistent with Arline were courts to credit lay testimony on matters of public health. See Arline,

480 U.S. at 287-88, 107 S.Ct. at 1130-31 (emphasizing the need to protect the handicapped from unfounded fears).

8. Dr. Bragdon cites a study reporting that compliance with the universal precautions would reduce needlestick exposures by only 62%. See Edward S. Wong et al., Are Universal Precautions Effective in Reducing the Number of Occupational Exposures Among Health Care Workers?, 265 Journal of the American Medical Association 1123, 1126 (1991). This statistic says nothing about the initial baseline degree of danger of treatment in the absence of the universal precautions and thus is impuissant to prove that a dentist using the CDC's precautions is directly threatened by treating an HIV-positive patient.

At this point, we have reviewed all the proof relevant to direct threat that Dr. Bragdon claims was available to him in September of 1994. Each piece of evidence is too speculative or too tangential (or, in some instances, both) to create a genuine issue of material fact. This ends our item-by-item explication of the record.

We next consider whether these eight proffers, in cumulation, possess greater probative force. This can occur when items of evidence, each insufficient in itself to prove a particular point, complement each other, like interlocking pieces of a jigsaw puzzle, in such a way that they together demonstrate some material fact. Thus, had Dr. Bragdon cited separate sources of evidence demonstrating (a) the likelihood of dental needlestick and (b) the likelihood of a dentist contracting HIV from a needlestick, these items together possibly would have been adequate to defeat summary judgment on the direct threat defense. Or, had he proffered several items of evidence that each showed a small risk to the health or safety of others, the aggregate effect of these items might have sufficed to prove a significant risk and thus to thwart summary judgment.

Despite the fact that Dr. Bragdon did not explicitly make a "cumulative proof" argument, we have spontaneously reviewed the record with this thought in mind. Having done so, we are satisfied that the evidentiary proffers canvassed above, insufficient in themselves, are likewise insufficient in combination to call into legitimate question the lower court's entry of summary judgment. In making this evaluation, we emphasize that, under the ADA, a service provider like Dr. Bragdon is not entitled to demand absolute safety; he can rely upon the direct threat defense only in response to significant risks. Here, Dr. Bragdon has failed to present *949 meaningfully probative evidence that treating Ms. Abbott would have posed a medically significant risk to his health or safety.

V. CONCLUSION

For the reasons indicated, we rule that Ms. Abbott's HIV positive status is a physical impairment which substantially interferes with her major life activity of reproduction, and that she is therefore disabled within the meaning of the ADA. Inasmuch as Dr. Bragdon has failed to produce sufficient evidence to establish a triable issue on his direct threat defense, the entry of summary judgment in Ms. Abbott's favor must stand.

Under ordinary circumstances, we would go no further. Here, however, we believe that more should be said. It is sometimes convenient to think of cases as involving conduct that may be categorized in terms of polar extremes: reasonable or unreasonable, praiseworthy or blameworthy, good or evil. But, given the complexities of the society in which we live, many decisions resist such facile classification into black-or-white dichotomies. Such cases are better characterized in varying shades of gray.

This is such an instance. The litigants' positions are understandable in human terms and impartial observers can empathize with both parties. Still, on the facts of record, the defendant's refusal to render routine dental care to an HIV-positive patient offends a duly enacted federal statute and thus cannot be tolerated by a court of law.

Although we do what we must, we are not blind to the difficulty of the choices that the ADA compels health-care professionals such as Dr. Bragdon to make. We also recognize that cases of this kind are necessarily fact-sensitive; had the patient required more invasive treatment or had the dentist proffered stronger evidence of a direct threat, the result may well have differed. In the same vein, presented with other facts and circumstances in a future case, perhaps reflecting dramatic improvements in medical science that substantially reduce the likelihood of transmitting HIV through reproduction, we might well reach a different conclusion than the one that we reach today. Such an ebb and flow is to be expected, because this is the very nature of the inquiry that the ADA mandates. We therefore caution future courts not to read our words more broadly than the context admits; our decision today eschews a blanket rule and instead demands case-by-case inquiry into a service provider's responsibilities to treat HIV-positive patients.

Affirmed.

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Defendants-Appellees,

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant.

ON REMAND FROM THE UNITED STATES SUPREME COURT

Before

Selya, Circuit Judge,
Aldrich, Senior Circuit Judge,
and Stahl, Circuit Judge.

ORDER OF COURT

Entered; August 14, 1998

Pursuant to the opinion of the United States Supreme Court, see Bragdon v. Abbott, 118 S. Ct. 2196 (1998), we direct the parties to file simultaneous supplemental briefs within thirty days of the date of this order, not to exceed twenty-five pages per side, devoted to the sole remaining issue in the case, namely, the Supreme Court's direction that we reconsider whether there was sufficient evidence to warrant summary judgment for the plaintiff on the issue of whether her proposed treatment "posed[d] a direct threat to the health or safety of others." 42 U.S.C. § 12182(b)(3). The supplemental briefs should address, inter alia, the parties' insights as to the import and admissibility of the 1993 CDC Dentistry Guidelines and the 1991 American

Dental Association Policy on HIV, referred to in both the Supreme Court's opinion, 118 S. Ct. At 2211-12, and our opinion, Abbott v. Bragdon, 107 F.3d 934, 945-46 (1st Cir. 1997). The briefs should also address whether there are any additional items in the record that bear on the question whether the evidence presented by (or favorable to) the defendant is sufficient to create a genuine issue of material fact as to direct threat.

The panel reserves, pending its receipt and consideration of the supplemental briefs, any determination as to whether further oral argument will aid the decisional process. If the panel answers that question affirmatively, oral argument will be scheduled as promptly as practicable. If the panel answers that question negatively, the court will decide the issue on the record (including the supplemental briefs).

By the Court:

Phoebe D. Morse, Clerk

By: JANICE M. O'NEIL
Chief Deputy Clerk

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Defendants-Appellees,

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant.

ON REMAND FROM THE UNITED STATES SUPREME COURT

MOTION FOR REMAND
(1) FOR FURTHER DISCOVERY AND DEVELOPMENT OF THE RECORD ON THE IMPORT AND ADMISSIBILITY OF THE 1991 AMERCIAN DENTAL ASSOCIATION POLICY ON HIV
AND
(2) FOR FURTHER TESTIMONY TO CLARIFY EXPERT OPINIONS AS TO INFORMATION AVAILABLE TO DENTISTS BEFORE SEPTEMBER 16, 1994

Background and Legal Argument

This case was briefed by the parties in the District Court several years ago for summary judgment purposes, and before then, discovery was conducted, on the assumption that the fact finder at trial would make a direct determination of whether or not there would be a direct threat/significant risk, *see* 42 U.S.C. § 12182(b)(3), to Dr. Bragdon or his staff in filling Ms. Abbott's cavity in his office. The assumption was that the fact finder would make a determination based upon expert testimony and the most up-to-date scientific and medical information. From Ms. Abbott's perspective, and as it turned out, the District

Court's, this included certain affidavits of Donald Marianos, D.D.S., M.P.H., of the Centers for Disease Control and Prevention ("CDC"), which expressed views that were not part of the public domain as of the date of Ms. Abbott's appointment with Dr. Bragdon, September 16, 1994. On the basis of Dr. Marianos' affidavits, and because Dr. Bragdon did not demonstrate that they were "medically unsupportable," the District Court concluded that treatment of Ms. Abbott in Dr. Bragdon's office "poses no direct threat to the health or safety of others."

On appeal, Dr. Bragdon argued that it was unfair and contrary to law to defer to affidavits of Dr. Marianos when they post-date Ms. Abbott's appointment and contain opinions absent and different from views expressed in official, published CDC materials. Dr. Bragdon therefore argued that the real question was whether, based on evidence available as of the date of the Ms. Abbott's appointment, an objectively reasonable dentist could have deemed the risk significant, even if reasonable experts could, did, or do disagree. In other words, Dr. Bragdon argued on appeal for a legal standard based on reasonable judgment and then-current medical knowledge. Under this standard, the fact finder would not have to determine whether or not there actually is a significant risk when filling a cavity. Instead, it would be enough for Dr. Bragdon to show that his risk assessment was reasonable based on then-current medical knowledge, even if reasonable experts can disagree.

This Court addressed this legal issue over Ms. Abbott's objection. The Court partially agreed with Dr. Bragdon, holding that as a general rule, health service providers should be judged based on sources of information available to them at the time treatment was refused, or in this case, deferred to a different setting. *Abbott v. Bragdon*, 107 F.3d 934, 944 (1st Cir. 1997). The Court disagreed with Dr. Bragdon on the issue of deference, holding that it is (apparently) not enough for him to show that reasonable experts could have disagreed. Instead, the Court said that he must overcome a continuing presumption of correctness

attached to the opinions of public health authorities. The Court then (apparently) cited as one such authority a 1991 American Dental Association (the "Association") policy statement on HIV. *Id.* at 945-46.

The United States Supreme Court vacated the judgment against Dr. Bragdon. Six justices expressed an opinion on whether Dr. Bragdon raised a genuine issue for trial. Four thought he did and that the case should be remanded for a trial (Rehnquist, C.J., O'Connor, Scalia, and Thomas, JJ.) Two thought he did not (Stevens, Breyer, JJ.) Three were uncertain (Kennedy, Souter, Ginsberg, JJ.). In order to defeat the four justices who would have simply remanded for a trial, Stevens and Breyer, JJ., joined with Kennedy, Souter, and Ginsberg, JJ. The case was thereby remanded to this Court instead of the District Court for trial.

Like this Court's opinion, the Supreme Court's opinion refers specifically to the Association's 1991 policy statement on HIV. Unlike this Court's opinion, the Supreme Court observes that the Association is not a public health authority, and states that "[w]ithout more information on the manner in which the American Dental Association formulated this Policy, we are unable to determine the Policy's value in evaluation whether petitioner's assessment of the risks was reasonable as a matter of law." This Court has accordingly asked the parties to address, *inter alia,* the import and admissibility of the Association's 1991 policy statement on HIV. *Abbott v. Bragdon,* No. 96-1643, Order of Court (1st Cir. Aug. 14, 1998).

The historical and epistemological origins of the Association's 1991 policy statement on HIV (e.g., who wrote it, based on what, if any, empirical research or review of the literature, was there a vote or an official minority view or contrary views expressed in committee or by some other process?) was not a subject of discovery. This was for several reasons.

First, the precise wording of the 1991 policy statement is not logically inconsistent with Dr. Bragdon's view that there would have been a significant risk in his office, as the term "significant" is used in the Americans with Disabilities Act.

Second, when discovery was in progress in 1995, the Association's 1991 policy statement (without citation to any underlying medical or scientific authority) seemed already somewhat outdated and of questionable relevance. The parties were focusing on the most up-to-date, original sources of information in medical and professional journals, and in deposing six expert witnesses (four for Ms. Abbott and two for Dr. Bragdon – counting Dr. Bragdon as an expert, which will still be in dispute if there ever is a trial) as to the basis and nature of their current opinions. No attempt was made to divide their opinions by date or by date of authority relied upon, and each expert cited as authority numerous materials dating both before and after Ms. Abbott's appointment. For example, although Dr. Molinari listed the Association's 1991 policy on HIV as one authority upon which he relied, he also listed approximately 150 other publications, most of which post-date the 1991 policy statement. In addition, the contemporaneous opinion of the American Association of Orthodontists was that in health care settings "scientific evidence indicates that HIV disease poses a direct threat and significant risk" and "much uncertainty surrounds what precautions, including barrier techniques, if any, can prevent the transmission of HIV disease." Comment No. 863 at 5 (April 23, 1991) in ADA Comment Book #1 for Public Inspection, available through Justice Department, disability rights division, 4th floor, 1425 New York Ave. N.W., Washington, D.C.

Under these circumstances, and according to the legal theory by which the case was proceeding, it did not make sense to explore through discover (e.g., by a deposition of one or more Association officials) the formulation of the Association's 1991 policy statement on HIV. This was especially true since the Association was not a party to the case, had not intervened, and

no expert witness purported to speak for the Association.

Now, however, the Association's 1991 policy statement is taking center stage. Not only has this Court asked Dr. Bragdon to address the concerns raised by the Supreme Court as to the formulation of the policy, but Dr. Bragdon has been recently informed by the Association that it intends to file an amicus brief against him in which it will purport to explain and defend its 1991 policy statement.

For many years there has been controversy within the Association regarding its policy on HIV. Many members disagreed with it in 1991, and many members continue to disagree based on scientific, medical, ethical, and public health policy concerns. However, in what is essentially an on-going political/professional/economic debate within a more or less democratically structured large professional organization, those who support the policy have prevailed.

However, nothing to this effect was developed through discovery in the record below for reasons already stated. In addition, the Association, as *amicus* for Ms. Abbott, is not likely to give an unbiased account of the formulation of its own policy (e.g., whether there was a minority committee report, or the like), and it is not likely to subject the scientific underpinnings of its policy to critical analysis. Without a remand for further development of the record in this regard, Dr. Bragdon is left with having to rebut assertions and arguments without knowing what they will be. For example, if the Association claims that its policy is based on a careful review of this or that article, how on earth is Dr. Bragdon supposed to point out the errors in inferences from that article, or point out conflicting articles, when he does not know which articles the Association will claim to rely on? If the Association claims that its staff conducted this or that form of independent research, how is Dr. Bragdon supposed to be able to expose weaknesses in methodology or bias in sampling? If there were minority committee reports, minutes of vigorous disputes as to the scientific basis of the

policy, earlier drafts of the policy which shed light on its application *vel non*, and so forth, how is Dr. Bragdon supposed to obtain them, or for that matter, even if he does, how are they to become part of the record? Forcing Dr. Bragdon to shoot in the dark outside the record on a matter that both this Court and the Supreme Court think may be central to this case cannot be what the Supreme Court had in mind when it observed that "full briefing directed at the [direct threat] issue would help place a complex factual record in proper perspective." *Bragdon v. Abbott*, 118 S. Ct. 2196, 2213 (1998). The Court should therefore remand for further development of the record through affidavits and discovery as to the historical origins and epistemological foundation of the Association's policy. Such discovery could efficiently proceed under the guidance, if necessary, of the District Court should any dispute arise as to the legitimate scope of the process.

In addition, the parties are being asked to brief the issue of how things stood as of September 16, 1994, whereas the expert reports and testimony developed in the District Court do not differentiate according to that date. Indeed, this Court previously ignored Dr. Kuvin's expert testimony on behalf of Dr. Bragdon because he relied partly on post-September 1994 authority in formulating his ultimate opinion that there is a direct threat. Yet Dr. Kuvin's opinion is plain that both before and after September 16, 1994 Dr. Bragdon could have reasonably assessed the risk as significant, and Dr. Kuvin's analysis of the risk is clearly relevant to whether there was medical or other objective evidence of a direct threat to dentists in September of 1994. Likewise, the Supreme Court observed that the "record does not disclose whether the expert testimony submitted by [Abbott] turned on evidence available in September 1994. See id., at 69-70 (expert testimony relied in part on materials published after September 1994)." *Bragdon*, 118 S. Ct. at 212. The Court should therefore remand to permit Dr. Kuvin to re-direct his testimony by pointing out explicitly the authorities and inferences therefrom that were available to dentists like Dr. Bragdon in September of 1994. To do otherwise would be less

than fair given the unique history of the case and the gravity of the matters at stake.

If the Court denies this motion, then at a bare minimum Dr. Bragdon deserved the right to file a response brief to any brief filed by the Association, or by Ms. Abbott or any one else, that is based directly or indirectly on any information or advice obtained from the Association with respect to its policy or matters relating thereto.

Dated: August 21, 1998

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No. 96-1643

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

SIDNEY ABBOTT, ET AL.,
Plaintiff-Appellees

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant

ON REMAND FROM THE UNITED STATES SUPREME COURT

BRIEF OF DEFENDANT/APPELLANT RANDON BRAGDON, D.M.D.

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PREFACE

"Efforts to clarify dentists' ethical obligations and to encourage dentists to treat patients with HIV infection with compassion may be commendable, but the question under the statute is one of statistical likelihood, not professional responsibility."

-- *Bragdon v. Abbott*, 118 S. Ct. 2196, 2212 (1998)

STATISTICAL SUMMARY OF THE ARGUMENT

1 in 66,667	Risk that a dentist will be infected with HIV by accidental needlestick when giving a single injection to an HIV- infected patient, using figures available in September 1994. [606a][1]
1 in 100,000	Risk of a fatal accident while skydiving in 1992. [592a, 596a]
6	Number of CDC-confirmed transmissions of HIV to dental patients during receipt of dental care from one HIV-infected Florida dentist, as of September 1994. [357a-379a]
0.8% to 8%	CDC official Dr. David Bell's 1991 calculation of the risk of an HIV-infected dentist transmitting HIV to at least one dental patient during seven years of practice due to an accidental sharps injury. [172a-181a]
"much greater"	Abbott's expert Dr. John Molinari's assessment in his 1991 book *Practical Infection Control in Dentistry* of the risk of accidental transmission

[1] ["[606a]", for example, refers to page 606a of this petition appendix.] *See infra* Parts II and V(C) for a detailed discussion of the scientific basis of this and other statistical calculations.

of HIV from patients to dentists compared to the risk of accidental transmission from dentists to patients. [190a-191a, 570a]

10% Dr. Kuvin's calculation of the risk of a dentist becoming infected with HIV by accidental needlestick during 30 years of practice if he gives an average of one injection to an HIV-infected patient each working day. [606a]

"Certainly superficial needlesticks have resulted in HIV transmission."

Response by CDC official Dr. David Bell during 1991 CDC open meeting to suggestion that some needlestick exposures to HIV may be trivial. [188a]

300% - 500% The amount by which the risk to a dentist from one needlestick exposure to HIV during his lifetime *exceeds* a level of risk OSHA deems clearly significant in the workplace. (*see infra* pp. [94a-98a]

"4 weeks (28 days) of both zidovudine 600 mg every day in divided doses (i.e. 300 mg twice a day, 200 mg three times a day, or 100 mg every 4 hours) and lamivudine 150 mg twice a day."

Current CDC recommended post-exposure drug prophylaxis ("PEP") for reducing risk of HIV infection after a "**superficial scratch**" (emphasis added) with a contaminated needle while working on an asymptomatic HIV-infected patient with high CD4 count. [201a-204a, 214a]

24%--36% Percentage of health care workers reporting subjective side effects (e.g., nausea, vomiting,

insomnia) causing them to discontinue PEP. [197a]

Unknown Amount by which PEP reduces the risk of HIV infection following percutaneous exposure to HIV. [193a]

"They're probably about the same."

Former CDC Associate Director for HIV/AIDS Dr. James Curran's 1989 assessment of the relative risk of contracting HIV as between one sexual contact with anal intercourse and one needlestick from an AIDS patient. [221a]

7 Number of CDC reported possible occupational transmissions of HIV to dental workers, including dentists, as of September 1994. CDC described them as dental workers infected with HIV who "have been investigated and are without identifiable [non-occupational] behavioral or transfusion risks" and who reported percutaneous or mucocutaneous occupational exposures to [HIV]." [224a]

62% Wong, et al.'s, March 1991 JAMA estimate based on their study data of the amount universal precautions should reduce the risk of occupational HIV transmission to health care workers. [633a]

0% The amount universal precautions should be expected to reduce the risk of occupational HIV transmission to health care workers based on the *statistical significance* of Wong, et al.'s study data, as clarified four months later in July 1991 edition of JAMA. [226a-227a]

"Most personal protective equipment is easily penetrated by needles."

> OSHA's current answer to the question "Why are universal precautions and personal protective equipment not adequate to protect the health care worker against needlestick injuries?" [236a]

Gloves "cannot prevent penetrating injuries due to needles or other sharp instruments" and "[d]uring dental procedures . . . trauma to health-care workers hands is common."

> CDC making essentially the same point in 1988. [466a]

"Reduction of risk factors in both professional and personal arenas remains the cornerstone in conscientious and responsible actions designed to guarantee absolute minimal risk of infection. The time, effort, and even dollars invested toward the pursuit of minimal risk is the most responsible option available to the health-care worker to provide both personal and patient confidence in establishing minimal disease risk."

> Abbott's expert Dr. John Molinari's advice to dentists in August 1993, Compend. Contin. Educ. Dent., vol. XIV, no. 8. [243a]

ARGUMENT

THE EVIDENCE IS SUFFICIENT TO CREATE A GENUINE ISSUE OF MATERIAL FACT AS TO DIRECT THREAT

It is undisputed that dentists risk becoming infected with HIV when they perform procedures that expose them to the

blood or bloody saliva of HIV-infected patients. *See, e.g.*, 56 Fed. Reg. at 64055, table VII (OSHA reporting 316,237 dental workers at risk of contracting HIV); *id.* at 64016-64022 (describing occupational transmission of HIV). Experts agree that the risk to an uninfected dentist while working on an HIV-infected patient is greater than the risk to an uninfected patient from an HIV-infected dentist. [191a, 581a, 246a] This assessment of the relative risks was repeatedly made known to dentists on or before September 16, 1994.[2] This is important because the existence or nonexistence of a direct threat must be determined from the standpoint of such dentists. *Bragdon*, 118 S.Ct. at 2210.

The dispute here is whether the identified and universally recognized risk to dentists is "significant." The "significance" of the risk to dentists depends on at least three factors: The severity of the harm risked, the quantitative likelihood (probability) of the harm's occurrence, and an ultimately qualitative assessment

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[2] *See* [191a] (referring to the 1991 first edition of *Practical Infection Control in Dentistry*); [570a] (Oct. 1993 article in J. of American Dental Assoc.); [253a] (Aug. 1993 article in Compend. Contin. Educ. Dent.).

of whether that probability is "significant" in light of the severity of the harm risked. In this case, the first factor is known and the latter two are in dispute.

I. Known Factor: HIV-Infection Is An Incurable Fatal.

HIV infection is an incurable fatal disease. *Bragdon*, 118 S. Ct. at 2203-2204. The severity of the risk (i.e., the potential harm) is a factor in determining whether a risk is significant. 28 CFR § 36.208(c); *School Bd. of Nassau County v. Arline*, 480 U.S. 273, 288 (1987). A large risk of a small harm may be insignificant, although a small risk of a great harm may be significant. There is no greater harm than loss of life. *See Doe v. Johnston*, 476 N.W.2d 28, 31 (Iowa 1991) (materiality of risk was properly a jury question when conflicting expert testimony put risk of contracting HIV between 1/100,000 and 1/1,000,000); *Carter v. Temple-Inland Forest Products Corp.*, 943 S.W.2d 221 (Tex. App. 1997) (reasonableness of party's fear of developing cancer was properly a jury question when the evidence was that the risk of developing cancer was between 1/100 to 1/500,000); *see also Montalvo v. Radcliffe*, No.

3:97cv487 (U.S. Dist. Ct. E.D. Va. Jan. 29, 1998) (finding that HIV-infected student posed direct threat to others in karate class where students frequently suffered minor cuts and nose bleeds); Richard Neustadt & Harvey Fineberg, *The Swine Flu Affair*, pp. 1, 69-73 (1978) (1/100,000 risk of developing a sometimes fatal paralysis from swine flu vaccine deemed significant by public health authorities when epidemic of swine flu did not materialize); Arthur Silverstein, *Pure Politics & Impure Science*, pp. 116-122 (1981) (reporting the same); Aff. of Donald E. Craven, ¶ 11 (Docket Doc. 62 filed 9/19/95) (reporting 1/50,000 to 1/100,000 risk of fatal reaction to penicillin and citing Erffmeyer, *Adverse Reactions to Penicillin*, 47 Ann. Int. Med. 288, 288 (1981), which describes that level of risk as a "significant health problem").

The fact that Dr. Bragdon is a dentist rather than a patient is legally irrelevant to the level of risk he may be required to take. As the Supreme Court held, "efforts to clarify dentists' ethical obligations and to encourage dentists to treat patients with HIV infection with compassion may be

commendable, but the question under the statute is one of statistical likelihood, not professional responsibility." *Bragdon*, 118 S. Ct. at 2212. In other words, the issue is Dr. Bragdon's legal, not moral or professional obligation, to put his and his assistants' lives in danger for the sake of another citizen in a non-emergency situation. The law does not force Dr. Bragdon and his assistants to take greater risks than patients or other citizens.[3]

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[3] Although the Supreme Court made it clear the issue is not professional obligation, Abbott may continue to malign Dr. Bragdon for shirking his duties as a dentist. To set the record straight, Dr. Bragdon did not deny dental care to anyone. He decided it was his professional duty to face the added risk (the "direct threat" in the most ordinary meaning of those words, if you have ever handled a needle while trying to inject someone in the mouth) of having an accidental sharps injury while working on a patient known to carry HIV, and he thought that the best way to minimize that risk and other risks consistent with the highest possible standard of care for his patient would be to perform the procedure using state-of-the-art infection control in the most aseptic environment available. In particular, and contrary to Abbott's assertions, Dr. Bragdon testified that when he offered to fill her cavity in a hospital, he believed that Eastern Maine Medical Center ("EMMC") "maintained [dental facilities] and general dentistry could be done at that facility without any problem" and EMMC would have been the "first place" he would have looked to if she had accepted his offer. [261a, 265a]. EMMC is located less than 1.2 miles from where Abbott lived in Brewer and less than 1.1 miles from where she worked. [584a] Dr. Bragdon's office is three times as far away.
Dr. Bragdon also testified that he would have considered using St. Joseph Hospital. [260a] St. Joseph is less than a mile from where Abbott worked. It was only after Abbott declined his offer that Dr. Bragdon learned the head of dental services at EMMC and St. Joseph was against the idea of general dentists working in the hospital. [262a-264a] Thereafter Dr.

II. First Disputed Factor: Quantification Of The Risk.

Dr. Bragdon's expert, Sanford Kuvin, M.S., M.D., D.T.M.&H, testified that a reasonable estimate of the risk that a dentist will accidentally contract HIV when giving a single intra-oral injection to an HIV-infected patient is 1 in 66,667. [606a] Dr. Kuvin further testified that if a dentist gives an average

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Bragdon applied at Downeast Community Hospital in Machias for dental privileges, but ironically that failed mostly because Abbott was suing him and the hospital was worried about its own potential legal exposure. [312a-314a] Abbott has nonetheless repeatedly accused Dr. Bragdon of having violating his ethical duty by *refusing* to treat her. He decided to treat her, and said he would treat her in a hospital, which he thought would be EMMC. She said she would "think about it and get back to him." [579a] Instead of responding to him, she sued him. In no way is it fair to depict this as a violation of Dr. Bragdon's ethical responsibility to treat. In addition, even health care providers specifically preferred by HIV-infected patients take additional precautions for themselves and their patients when performing otherwise routine procedures. *See* Confidential Document lodged July 18, 1997 with Supreme Court ("Confiden. Doc."). Because Abbott informed Dr. Bragdon that she had been diagnosed with HIV 8 years earlier [259a], and because most HIV-infected persons' immune systems decline substantially by then even without visible symptoms, *Bragdon*, 118 S. Ct. at 2204, it was especially appropriate for Dr. Bragdon, for the sake of a new patient about whom he knew next to nothing, to defer treatment to an aseptic hospital environment, after consultation with her physician, before he started injecting and cutting her gum tissue. *See* Confid. Doc. and [273a-299a] Dr. Bragdon has not focused on this in his summary judgment briefs because of page constraints. The fact remains, however, that experts believed prior to 1994 that even before an AIDS diagnosis, the longer a person is infected, the more infectious is their blood with respect to HIV, and the more likely the person may have active TB (or may be susceptible to or transmit other potentially serious infections which put themselves and dentists at risk during invasive and aerosol generating procedures) without realizing it. [603a-605a, 489a-492a]

of one intra-oral injection each working day to an HIV-infected patient for thirty years, the dentist has a 1 in 10 risk of accidentally contracting HIV during that lifetime of practice. [606a]

Dr. Kuvin is an expert in infectious diseases and holds a Master's Degree with Distinction in Medical Microbiology. [572a] Abbott has never challenged his qualifications as an expert or the admissibility of his testimony under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). Dr. Kuvin's calculation of the risk of contracting HIV per injection given to an HIV-infected patient (1 in 66,667) follows by mathematical necessity as the product of (A) dentists' rate of accidental needlestick injuries (1.5 needlesticks per 100 procedures), (B) the likelihood of contracting HIV following an HIV-contaminated needlestick injury (3 in 1000), and (C) the likelihood that an accidental needlestick while working on an HIV-infected patient will occur with a contaminated needle (1 in 3). [606a] Dr. Kuvin's calculation of the risk of contracting HIV during 30 years of practice giving an average of 235

injections per year to HIV-infected patients follows by mathematical necessity from the risk per injection and the number of injections. [606a, 611a-614a] Dr. Kuvin's estimates are scientifically based and create a genuine issue for trial as to the quantification of the risk. *Cf. Abbott v. Bragdon*, 107 F.3d 934, 948 (1st Cir. 1997) (evidence of the likelihood of dental needlestick and of contracting HIV from a needlestick may raise genuine issue of fact).

A. Rate of Needlestick Injuries. Dr. Kuvin used 1.5 needlestick injuries per 100 injections as a reasonable estimate of the likelihood of a dentist suffering a needlestick injury. [606a] United States' expert Dr. Greenspan reported this figure in her book *Aids and the Mouth*, which was published in 1990 and available to dentists in 1994. [316a] Dr. Greenspan testified in her deposition she believed the figure was reliable and not substantially different from that reported in later surveys. [248a-249a] Dr. Kuvin, on behalf of Dr. Bragdon, is entitled to rely on this figure at summary judgment. If plaintiff Abbott believes

other figures produce better estimates, she should present that testimony at trial and allow the fact finder to decide.

B. Rate of HIV Infection After Needlestick Exposure. Dr. Kuvin used 0.3% (3 in 1000) as a reasonable estimate of a dentist's rate of infection with HIV after a percutaneous exposure to HIV-infected blood by a needlestick injury. [606a] This estimated rate of infection is 17% *lower* than the figure reported as an average for all percutaneous exposures to HIV-infected blood by health care workers, including dentists, in a leading pre-September 1994 report, Tokars, et al., *Surveillance of HIV Infection and Zidovudine Use Among Healthcare Workers With Occupational Exposure to HIV Infected Blood*, Ann. Intern. Med., vol. 118, no. 12, p. 913 (June 1993). [318a-331a] The group of health care workers studied in the Tokars report included dentists. The report combines "physicians and dentists" into a *single* category for the purpose of specifying the categories of health care workers in the study. *Id.* p. 914 [321a] This supports Dr. Kuvin's use of 0.3% in his calculations of the risk to Dr. Bragdon.

In addition, in 1995 articles written *for dentists* to inform them of the risks of HIV, authors reported rates of HIV infection ranging from 0.42% to 0.3%. *See* Siew, et al., *Percutaneous Injuries in Practicing Dentists*, J. Am. Dental Assoc., vol. 126, p. 1227 (Sept. 1995) [333a]; Gooch, et al., *Percutaneous Exposures to HIV-Infected Blood Among Dental Workers Enrolled in the CDC Needlestick Study*, J. Am. Dental. Assoc., vol. 126, p. 1237 (Sept. 1995) [336a] The studies and data upon which these 1995 reports are based (including the higher figure of 0.42%) were available before September 16, 1994, as was OSHA's assessment of the HIV risk from needlesticks, which relies on a still higher reported infection rate of 0.47% following percutaneous exposure to HIV-infected blood. *See* 56 Fed. Reg. at 64032-64033. All of these objective sources further support Dr. Kuvin's use of 0.3% as a reasonable infection rate

for dentists as of September 1994.[4] If plaintiff Abbott believes other sources of information as of September 1994 support the use of a different infection rate, she should present that testimony at trial, and it will be up to the fact finder to decide.

In addition, objectively reasonable dentists in 1994 would have concluded from these reliable sources that just *one* accidental needlestick exposure to HIV-infected blood during a dentist's entire lifetime of practice puts him at 3 to 5 times the level of risk that OSHA and the Supreme Court deem "significant." Specifically, in the so-called "Benzene case," a plurality of the Supreme Court stated the obvious, namely, that if

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[4] Prior to 1994 some hypothesized that the HIV infection rate might be lower for solid-bore suture needles than for hollow-bore needles used for injections, or might be lower if the health care worker wore gloves (since passage of the sharp object through glove material might result in less blood exposure). *E.g.*, [177a] As of September 1994, there was insufficient scientific evidence to confirm that hypothesis, and it now appears that the glove theory is incorrect, and that regardless of the type of needle used, two significant mutually independent factors in infection are whether there is any visible patient's blood on the device or whether the health care worker sustains a deep injury, even without visible blood on the device. [663a-665a] ("use of gloves" was one of the variables examined to see if it had an impact on prevention, but no significant impact was found)) Based on Dr. Bragdon's own experience, and on published studies, there is often visible patient's blood on the device with which a dentist is accidentally injured. [340a] Dr. Kuvin's use of the 0.3% figure is therefore plainly within an acceptable range for estimating the risk to Dr. Bragdon.

the odds are 1 in a 1000 that exposure to a substance in the workplace will be fatal, a reasonable person could deem the risk significant. *Industrial Union Dep't, AFL-CIO v. American Petroleum Institute*, 448 U.S. 607, 655 (1980). Relying on this 1 in a 1000 standard for significant risk, OSHA found in December 1990 that a study published by Klein, et al., in the New England Journal of Medicine, suggests approximately that same significant level of risk (i.e., 1 in a 1000) for dentists of contracting HIV. *See* 56 Fed. Reg. at 64092. In effect, OSHA considered patients in general (HIV-infected and uninfected alike) as the "substance in the workplace" to which dentists are exposed. Because the Klein study found one likely occupational transmission of HIV among 1,309 dentists practicing among the general population, this equated with approximately 1 fatality per 1000 dentists exposed to a "substance" in the workplace, which OSHA deemed significant. [355a] The Klein study also reported that for dentists practicing in locations with a large number of reported AIDS cases, there appeared to be one likely occupational transmission of HIV among 523 dentists. [356a]

Finally, if one looks at the problem in terms of exposures to needlestick injuries, rather than exposures to patient pools, the risk of contracting HIV for a dentist who has just *one* accidental needlestick exposure to HIV during an entire lifetime of practice is 3 to 5 in a 1000 (i.e., 0.3% to 0.5%), which is 3 to 5 times OSHA's 1 in a 1000 standard for "significant risk." This high rate of HIV infection following percutaneous exposures to HIV-infected blood (3 to 5 times a rate deemed clearly significant by OSHA) explains why CDC recommends costly and painful post-exposure chemoprophylaxis ("PEP") for dentists who receive even superficial scratches from HIV-contaminated needles. [195a-197a, 201a-204a] *Cf. Hartwig v. Oregon Trail Eye Clinic*, 580 N.W.2d 86, 94, 254 Neb. 777 (1998) (survey of cases permitting recovery for mental anguish resulting from fear of testing positive for HIV after accidental needlesticks. and holding that "[w]e cannot say, as a matter of law, that one's fear of testing HIV positive . . . is unreasonable when one suffers an injury in a manner by which medical science has concluded that HIV could be communicated").

C. Rate of Needlestick Injuries Occurring With Contaminated Needles. Dr. Kuvin used 1 in 3 as his estimate of the rate at which a dentist is exposed to a contaminated needle when he suffers a needlestick injury. [606a] In other words, Dr. Kuvin estimated that 2 out of 3 times a dentist accidentally sticks himself with a needle while working on a patient, the needle is *not* contaminated, i.e., the needle was not yet used to inject the patient and was not previously in contact with the patient's blood or saliva after the patient bled.[5] This 1 in 3 "contaminated needle rate" used by Dr. Kuvin in his calculation of the risk to dentists per injection is analogous to the 32% (i.e.,

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[5] The saliva of HIV-infected persons contain HIV, but saliva is not normally considered an "Other Potentially Infectious Material" with respect to HIV. The single exception is dentistry, where OSHA decided to follow the CDC in treating saliva from dental patients as infective, since bleeding is so common in even the most routine dental procedures. [460a] According to OSHA guidelines, Other Potentially Infectious Materials include "saliva in dental procedures." 56 Fed. Reg. at 64102; *id.* at 64103 ("[w]hile universal precautions do not generally apply to saliva, exception is made in the case of saliva in dentistry"). Standard textbooks for dental workers therefore teach that "[d]uring dental treatment, saliva is *always* considered to be contaminated with blood. This is true even when blood is not visible in the saliva." [520a-521a] Since OSHA, CDC, and textbooks for dental workers all treat *all* saliva during dental procedures as potentially infectious for HIV, Dr. Kuvin is obviously entitled to treat a needle as contaminated if it has contacted the patient's saliva after the patient bled during a dental procedure.

just under 1 in 3) re-contact rate used by CDC official Dr. Bell in his calculation of the quantitative likelihood of the risk of transmission of HIV from surgeons or dentists to patients. [176a, 180a] The re-contact rate is the rate at which a sharp object causing an injury to a surgeon or a dentist re-contacts the patient's wound. Dr. Kuvin's use of 1 in 3 as a contaminated needle rate in his calculations is a conservative (i.e., low) estimate based on common experience, reported studies of the nature of needlestick injuries among health care workers, and the transmissibility of HIV.

An accidental needlestick with a needle can occur before, during, between, or after an intra-oral injection. [577a ¶ 7] Common sense suggests that even if a dentist is more clumsy than most, it would be unusual for him to stick himself with a needle before he even gets it to the mouth of the patient. Instead, many accidents will occur when or after the needle makes contact with the patient, causing the patient to flinch or bite down. As one study reported, "another [accidental needlestick with an HIV contaminated needle] occurred when a patient

suddenly grabbed the dentist's hand during injection of local anesthetic" [339a] Empirical studies of the nature of needlestick injuries among health care workers support the common sense assumption that *most* accidental needlesticks occur after the needle has been used on the patient. *See* 56 Fed. Reg. at 64033 (reporting Jagger study that 70% of needlestick injuries occurred after use but before disposal of the device).

Furthermore, studies published in 1990 and 1992 indicated that even *visually clean needles* can transmit HIV. *See* Chitwood, et al., *HIV Seropositivity of Needles from Shooting Galleries in South Florida*, Am. J. of Public Health, vol. 80, no. 2, p. 151 (Feb. 1990) ("selection of a needle without visible blood was no assurance of safety" since "even the visually 'clean' needles may result in transmission of infection") [380a, 386a]; *cf.* David Ho, *Quantitation of HIV-1 In Vivo*, HIV Advances in Research and Therapy, vol. 2, no. 3 (Oct. 1992) (significant tissue-culture—infective doses of HIV-1 can be found in micro-liters of blood) [394a-395a]. The fact that even visually clean needles can transmit HIV supports Dr. Kuvin's

assumption that any injury to the dentist with a needle after it has been injected into an HIV-positive patient, or after it has contacted the patients' saliva following any amount of bleeding, is a life threatening event. In addition, according to a study published in 1995, but based on data available before December 1994, 6 of 19 sharps injuries suffered by dentists were from instruments with visible patient's blood on the device. [340a] In contrast to whether Bendectin causes birth defects, *Daubert*, 509 U.S. 579, or whether exposure to PCBs promote the development of lung cancer, *General Elec. Co. v. Joiner*, 118 S. Ct. 512 (1997), we know for certain that when dentists accidentally stick themselves, the instruments in question are often contaminated with the source patient's blood in amounts sufficient to transmit HIV, even in the absence of "visible" contamination. This fact, plus empirical evidence that more than half of all needlesticks happen after needles have been used on patients, *see* 56 Fed. Reg. at 64033, means that Dr. Kuvin's working assumption is quite modest that only 33% of the needles

with which dentists stick themselves are contaminated with blood or other potentially infectious materials.

D. Dr. Kuvin's Calculations Underestimate The Risk As Applied To Dr. Bragdon Because His Calculations Rely On A Reported Average Rate of Needlestick Injuries For All Dentists Rather Than The Observed Rate Of Sharps Injuries For Dr. Bragdon. Different dentists suffer significantly different rates of percutaneous injuries. For example, according to surveys cited by the CDC in May 1992, 32% of dentists in one study reported 2 or more sharps injuries (i.e., needlesticks and other sharp instruments) per month. [372a] Three percent of the dentists reported more than 15 such injuries per month. (*Id.*) Testimony from Dr. Bragdon suggests that he falls closer to the range of the 3% of dentists who averaged more than 15 such injuries per month, since in his experience he often suffers several each week.[6] [303a-304a] Because the issue is the risk

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[6] Because there were approximately 175,000 dentists in the United States, if only 1% of them suffered more than 15 injuries per month, that would be over 1,700 dentists in the country reporting rates of accidental sharps injuries similar to and higher than Dr. Bragdon. *See* Bureau of Economic and Behavioral Research, Distribution of dentists in the U.S. by region and state—1987.

that Abbott posed to Dr. Bragdon not just from an accidental needlestick concomitant to a single injection, but the risk from any sharps injury or other mode of HIV transmission, Dr. Kuvin could have relied on the observed rate of all sharps injuries for Dr. Bragdon, which is much higher than the 1.5/100 rate of needlestick injuries that he used in his calculation of a 1 in 66,667 risk per injection for all dentists. The result is that the actual risk to Dr. Bragdon per procedure performed with a sharp instrument on an HIV-infected patient is greater than 1 in 66,667.

This Court previously indicated that Dr. Bragdon's sworn deposition testimony as to how often he injures himself might not be probative of how often he injures himself. *Abbott*, 107 F.3d at 948. It is difficult to imagine how a statistical average for all or some dentists could be more probative on how often Dr. Bragdon cuts himself than Dr. Bragdon's own direct observations of what has happened to him during his career. *Cf. United States v. DiMarzo*, 80 F.3d 656, 659-660 (1st Cir. 1996) (*Daubert* not implicated in testimony expressing neither a lay

nor an expert opinion, but simply witness's personal experience relating to a subject bearing directly on the appropriateness of a jury inference). In addition, "[c]redibility determinations . . . are jury functions, not those of a judge [at the summary judgment stage]," and the "evidence of the nonmovant is to be believed." *Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 255 (1986). This means that just as the Court was not permitted to doubt Abbott's sincerity as to her stated reason for not having children, *Abbott*, 107 F.3d at 942, the Court is not permitted to doubt Dr. Bragdon's sincerity as to how often he injures himself, especially since there is objective evidence that many other dentists sustain injuries at rates similar or higher than those reported by Dr. Bragdon. [372a] (3% of dentists reporting more than 15 injuries a week) [352a] table 4 (dentists reporting between 1 and 600 accidental inoculations within one year). The wide variation in the rate of sharps injuries among dentists may also help to explain controversy among them as to the significance of the risk of working on HIV-infected patients, since it could be hard for a dentist who rarely cuts himself to

imagine what it is like for the thousands of dentists near the other end of the statistical curve.

E. Dr. Bell's Calculations of The Risk of Transmission of HIV To Patients Verify Dr. Kuvin's Calculations of The Risk of Transmission of HIV To Dentists.

During a 1991 CDC open forum, CDC official Dr. Bell calculated that the probability that an HIV-infected surgeon will transmit HIV to at least one patient due to an accidental sharps injury during 7 years of practice (assuming 500 procedures per year) is between 8.0% (using 0.3% as rate of HIV infection after percutaneous exposure) and 0.8% (using 0.03% as the rate of HIV infection after percutaneous exposure). [176a-178a] Dr. Bell's calculation is discussed at *Doe v. University of Md. Med. System Corp.*, 50 F.3d 1261, 1263 (4th Cir. 1995). Dr. Bell chose 7 years because that is an estimate of how long an average HIV-infected doctor may be expected to practice before death or debilitating symptoms due to his HIV infection. Dr. Bell's estimate of the risk of transmission of HIV by an HIV-infected surgeon to a single patient ranged from 1 in 42,000 (using the

0.3% infectivity rate) to 1 in 417,000 (using 0.03%). *Id.* Dr. Bell arrived at nearly the same figures for dentists using the same re-contact rate (32%), the same range of HIV-infectivity rates (0.03% to 0.3%), and 7 years of practice. The only difference was the number of procedures performed by dentists per year (3000 for dentists instead of 500 for surgeons) and the rate of sharps injuries (0.4 per 100 procedures for dentists instead of 2.5 per 100 procedures for surgeons). When factored into the calculation, the 0.4 injuries per 100 procedures times 3000 procedures per year for dentists results in 12 injuries per year, while the 2.5 injuries per 100 procedures times 500 procedures per year for surgeons results in 12.5 injuries per year. [176a-180a] Thus the risk to dental patients over 7 years from dentists is virtually identical to the risk from surgeons, while the risk to any one dental patient is less than any one surgery patient if the 0.4 injuries per 100 procedure estimate is correct for dentists. However, as we know, the 0.4 in 100 estimate may be too low both as a statistical estimate (since, e.g., Greenspan reports 1.5 in 100 for dentists—[316a], and too low for many practicing

dentists (since, e.g., 3% of dentists in one study reported more than 15 injuries per month. [372a].

These are Dr. Bell's estimates for CDC of the risk of dentist-to-patient HIV transmission, whereas this case concerns the best available estimates in 1994 of patient-to-dentist transmission. Dr. Bell's dentist-to-patient estimates are nonetheless relevant because they set a baseline level of risk against which to measure the objective validity of Dr. Kuvin's calculations. Dr. Bell's calculations set a baseline because Abbott's experts concede the risk to health care workers, including dentists, is greater than the risk to their patients.[7]

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[7] Experts opined as of 1994 that the risk to health care workers, such as dentists, is greater, in part because so many more documented and possible HIV transmissions to health care workers were reported, namely, 130 [224a], than transmissions to patients (only 6, all of which were to dental patients) [375a]. One thing that could account for this is that the contaminated instrument rate could be much higher than the re-contact rate, meaning it is more likely a health care worker will cut himself with a contaminated instrument than re-contact a patient with an instrument after he cuts himself. The Jagger study cited at 56 Fed. Reg. at 64033 (70% of injuries occur after instrument is used) supports this theory. For health care workers other than dentists and surgeons, it would be an odd circumstance for them to cut themselves and then use the instrument on the patient. In addition, health care workers are often exposed to visible amounts of patients' blood, whereas even if the health care worker draws blood when he sticks himself, it seems less likely that blood will contact the patient. *See* [190a-191a].

[191a, 246a, 570a, 581a] This means that a calculation of the likelihood of dentists contracting HIV, if it accords with the best available evidence for dentists at the time, should produce results that are comparable with or higher than Dr. Bell's estimates of the risk to dental patients. And this is exactly the case with Dr. Kuvin's calculations. Dr. Kuvin calculated that the risk to Dr. Bragdon of contracting HIV giving an intra-oral injection to an HIV-infected patient is 1 in 66,667 (i.e., 1/3 x .3% x 1.5/100). This by itself is comparable to (in fact, lower than) the figure reached by Dr. Bell for the risk to a single surgical patient (1 in 42,000, assuming a 0.3% infectivity rate, a 32% re-contact rate, and 2.5 sharps injuries per 100 procedures). The only difference in these two calculations is the rate of sharps injuries, with Dr. Kuvin using Greenspan's lower 1.5 in 100 figure for dentists.

In addition, using his 1 in 66,667 figure as an estimate of the risk to a dentist of contracting HIV when performing a single injection on an HIV-infected patient, Dr. Kuvin asked a statistician to calculate the likelihood that a dentist would receive at least one fatal needlestick injury over a lifetime of

practice (30 years) assuming the dentist gives 235 injections each year (about one each working day) to an HIV-infected patient. [606a] The result was that the dentist would have an approximately 10% risk of becoming infected with HIV on at least one occasion. (*Id.*) A 10% percent risk over a lifetime of practice (30 years) for an uninfected dentist is slightly higher than Dr. Bell's 8% risk over seven years of practice for uninfected dental patients. But this is exactly what one would expect given that the risk to dentists is greater than to the patients. As an order-of-magnitude estimate, Dr. Kuvin's calculation of the statistical risk to dentists checks out perfectly with analogous calculations put forth by public health officials based on the best objective evidence available to dentists in 1994. Furthermore, if "the best objective evidence" available to Dr. Bragdon of the risk to Dr. Bragdon includes his own direct observations of how often *he* cuts himself, which it should, then the level of risk to him is considerably higher than Dr. Kuvin's estimate.

No-one is suggesting that Dr. Bragdon gives an average of one injection per working day to an HIV-infected patient (although many dentists in some urban areas probably do). The point (which Abbott has steadfastly pretended not to see) is to test Dr. Kuvin's estimate of a 1 in 66,667 risk for a single injection by using that estimate to compare results with Dr. Bell's results when viewed from the perspective of numerous opportunities to transmit the disease, rather than a single opportunity. Dr. Bell's estimate of 500 procedures per year for a surgeon functions mathematically in these models in the same way as Dr. Kuvin's use of 235 injections per year. The difference is that while the average HIV-infected dentist or surgeon poses an approximately 0.8% to 8% risk of passing HIV at some point to a patient during 7 years of practice (using Dr. Bell's figures), only those dentists or surgeons who perform numerous procedures on HIV-infected patients have similarly high risks (i.e., 1% to 10% or higher) of acquiring HIV from patients. This, however, does not impugn the 1 in 66,667 figure as the risk per single injection. On the contrary, it lends strong

support for it, especially for considerations of summary judgment.

F. The Law Does Not Require Dr. Bragdon To Produce Evidence That Is Better Than The Best. Although the law might demand of Dr. Bragdon that he act based on the best available objective or medical information at the time, it does not demand that he act on evidence that is better than the best. The studies and modes of reasoning presented above and relied on by Dr. Bell and Dr. Kuvin operate at a level of precision and generality appropriate for experts in infectious diseases given the state of scientific knowledge in 1994, and given certain insuperable obstacles to definitive work in medical science (e.g., the inability purposely to expose subjects to known dosages of HIV on contaminated needles, and to keep them confined for eight months, to make sure they have no other exposure to HIV, in order to arrive at more precise infectivity rates). Empirical science is not a set of proven facts, but efforts to think logically about observable phenomena and to test proposed explanatory principles by what further observations are

available or may become available through experimentation. In the context of this case, Dr. Kuvin's and Dr. Bell's calculations of the apparent order of magnitude of the risks are scientific because they are grounded in the methods and procedures of science based on available data at the time. [8] OSHA likewise relied on this method of analysis to estimate the probability among health care workers of at least one HIV infection due to needlestick exposure to HIV-infected blood. *See* 56 Fed. Reg. at 64033, table V-10. The calculations of risk in these contexts are scientific, among other reasons, precisely because they are offered as estimates, and not as proven figures.[9]

Depending on the area of science, something like "scientific certainty" is approachable (e.g., in basic chemistry), but most of "good science" achieves nothing close to "scientific

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[8] *See Daubert*, 113 S. Ct. at 2795; *see also* Brief of the Carnegie Commission on Science, Technology, and Government as Amicus Curiae in Support of Neither Party, in *Daubert*, 113 S. Ct. 2786.

[9] *See* [180a, 184a, 187a] (Dr. Bell reiterating that his model has important limitations that "may result in over- or underestimates"; Dr. Howard acknowledging that Dr. Bell's assessment model is "certainly a good first attempt"; Dr. Curran defending Dr. Bell's estimate as a way of putting the risk into "logical bounds"); *see also* [606a] (Dr. Kuvin explaining that because of limited data, the best that can be done are "some fairly reasonable estimates").

certainty." [504a] ("Definitive results are hard to get in science. Usually, the evidence is circumstantial.") "Scientific certainty" has never been required for public health officials to act. *Cf. Industrial Union*, 448 U.S. at 656 (OSHA may regulate based on "the best available evidence," which need not be "anything approaching scientific certainty"). Scientific certainty has never been required for the practice of medicine, i.e., for the exercise of professional judgment in diagnosing and treating disease, and in attempting to prevent its transmission. *E.g.*, [193a] (CDC recommends PEP to reduce significant risk of infection although "[t]here is little information with which to assess the efficacy of PEP in humans"); [502a] (physician explaining the difficulty of making treatment decisions in "uncharted territory"). To suddenly require an individual defendant in private civil litigation to produce evidence at summary judgment superior in quality and certainty than the best available evidence and methods of analysis relied on by others in the field, including plaintiff's experts, OSHA, and CDC, is to demand far better than the best. There is therefore nothing that may properly or

thoughtfully be discarded as "unscientific" or "conjecture" in Dr. Kuvin's testimony and Dr. Bragdon's presentation of the evidence. *Cf. Joiner*, 118 S. Ct. at 520 (Breyer, J. concurring) (judges are sometimes asked "to make subtle and sophisticated determinations about scientific methodology and its relation to the conclusions an expert witness seeks to offer—particularly when a case arises in an area where the science itself is tentative or uncertain."); *Mitchell v. U.S.*, 141 F.3d 8, 15-17 (1st Cir. 1998) (inability to quantify risk and weak factual underpinnings go to the weight and credibility of expert's testimony at trial, not its admissibility).

Furthermore, Abbott did not challenge Dr. Kuvin's testimony under *Daubert* below. For that reason alone, she is prohibited from doing so on appeal. *See Cortes-Irizarry v. Corporacion Insular De Seguros*, 111 F.3d 184, 188-189 (1st Cir. 1997) (there are few, if any cases in which an appellate court should employ *Daubert* where there has been no development of the issue below, and courts must be cautious not to exclude debatable scientific evidence without affording the proponent

adequate opportunity in a trial-like setting to defend its admissibility).

G. Abbott Disputes Dr. Kuvin's Quantitative Assessment of the Risk. Abbott challenges Dr. Kuvin's calculations. Her principal argument is that because CDC in June 1994 reported just 7 possible occupational transmissions of HIV to dental workers, and no documented transmission of HIV to a dental worker among 42 documented transmissions to other health care workers [224a], the only valid scientific conclusion is that the risk to dentists is infinitesimal, or at any rate, much less than Dr. Kuvin's calculation. On scientific grounds, this argument is invalid for reasons set forth below, *see infra* Part V. It is noteworthy, however, that Abbott's argument (lack of documented cases to fit the model) is essentially the same argument made at the 1991 CDC open meeting by an angry participant from the floor against Dr. Bell's estimates. [186a-187a][10] Although CDC official Dr. Curran's response was

[10] The angry participant refers to 3 transmissions in one office. The participant is referring to what were at that time reported transmissions of HIV from a Florida dentist to three of his patients. In May 1992, CDC

polite, he did not think Dr. Bell's risk assessment models were thereby refuted. [180a, 186a-187a] There is therefore a genuine and material dispute of fact as to the quantification of the risk.

III. Second Disputed Factor: Qualitative Significance Of The Risk.

The second disputed factor is the qualitative significance of the risk. Although Abbott does not now argue the point, she will probably argue if there is a trial that even if the risk is 1 in 66,667 per injection, or 1 in 10 over a lifetime of practice treating HIV-positive patients, that is not a qualitatively "significant" risk, even when the risk is a horrible, incurable disease. *Cf. Industrial Union*, 448 U.S. at 655 n. 62 (majority agreeing that while purely factual determinations can define a

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updated that figure to five patients [357a], and in May 1993, CDC concluding that a sixth patient also contracted HIV from the dentist [375a]. Unlike the previous five patients, "this [sixth] patient had neither dental extractions nor root canal therapy." [378a] Other than X-rays and a cleaning, there was only a single surface restorative filling [377a], like the "routine procedure" at issue in this case.

risk in some statistical way, a judgment of "significance" is based on other factors). Abbott will probably point to statistics about everyday risks such as highway driving and what not, and Dr. Bragdon will point to statistics, such as the risk of fatal accidents while skydiving, or of having died if called to serve in the Viet Nam conflict, as well as examples in recent history where public health officials have deemed similarly low risks significant. Here the situation is not unlike an ordinary negligence case in which one expert re-constructs the crash site and says the defendant was going at least fifteen miles over the speed limit, and the other expert says it was at most five miles over the speed limit on a clear day. Even if the jury believes the latter expert, the jury would still be permitted to find negligence, or the absence of negligence. This is the classic qualitative, evaluative function of the jury. This is the sort of finding that the jury would be called on to make in deciding qualitatively whether, "from the standpoint of the person [i.e., Dr. Bragdon] who refuse[d] the [in-office] treatment," *Bragdon*, 118 S. Ct. at 2210, was the risk "significant" based on "medical or other

objective evidence," *id.*; *cf. United States v. Rule Industries, Inc.*, 878 F.2d 535, 541 (1st Cir. 1989) (describing instances in which juries are called upon to make evaluative applications of law to fact even in the absence of disputed issues of historical fact).[11]

IV. NEITHER THE 1993 CDC DENTISTRY GUIDELINES (THE "GUIDELINES"), NOR ANY OTHER PUBLIC HEALTH AUTHORITY ON RECORD BEFORE SEPTEMBER 16, 1994, STATED EXPLICITLY OR IMPLICITLY THERE IS NO DIRECT THREAT TO DENTISTS; THEREFORE, THE RULE THAT THE VIEWS OF PUBLIC HEALTH AUTHORITIES ARE OF SPECIAL WEIGHT IS NOT APPLICABLE AGAINST DR. BRAGDON IN THIS CASE.

A. The Guidelines Do Not Assess The Level Of Risk.

The Supreme Court is correct that the Guidelines [527a-547a] "do not assess the level of risk." *Bragdon*, 118 S. Ct. at 2211. Instead, the Guidelines state that their implementation "should reduce the risk of transmission of infectious diseases caused by bloodborne pathogens such as HBV and HIV." [529a] No attempt is made quantitatively or qualitatively to assess the level

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[11] Dr. Bragdon actually believes that no reasonable person could deem the risk insignificant based on the available medical and other objective evidence. He therefore renews his request for summary judgment in his favor.

of risk of transmission of HIV to dentists either before or after implementation of the Guidelines.[12]

In addition, "should," as in "should reduce the risk of transmission," meant "might not," since experts knew then, as they know today, there is no scientific support for the view that universal precautions lower the risk of transmitting HIV. For example, when Senator Daniel Moynihan inquired of CDC on behalf of a dentist in 1995 as to the efficacy of taking "universal

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[12] Under the bold heading "**CONFIRMED TRANSMISSION OF HBV AND HIV IN DENTISTRY**," the Guidelines say that "the possibility of transmitting bloodborne infections from DHCWs to patients is considered to be small." [530a] This statement is about the quantitative likelihood of transmission of HIV from dentists to patients, not the qualitative significance of the risk. For example, if a patient were told by an HIV-infected dentist that the risk is "small" of the patient contracting HIV during a procedure, it would not be irrational for the patient to ask "How small?" before deciding whether the risk is significant. *Ipso facto*, the statement does not assess the significance of the risk to dentists. To the extent the statement bears at all on the significance of the risk to dentists, it tends to support Dr. Bragdon. This is because Abbott's expert Dr. Molinari conceded that the risk to dentists like Dr. Bragdon are much greater. Whatever "much greater" than "small" may mean, it hardly seems likely it is "infinitesimal" or "trivial" as Abbott maintains, for otherwise CDC would have characterized the risk to patients as infinitesimal, negligible, or trivial. Objectively reasonable dentists in 1994 understood the difference between "small" and "trivial" when the consequence is death, they understood what words CDC chose, and they understood that experts told them the risks to them were greater than to their patients. For summary judgment purposes, this alone should be enough to defeat Abbott's contention that Dr. Bragdon could only have been acting out of ignorance and irrational fears.

precautions," CDC referred Senator Moynihan to "the two latest articles" on the subject. [621a] In September 1994, Dr. Bragdon could not have known what CDC's opinion was in 1995 as to the "latest articles" on the subject. However, since both articles endorsed by CDC were published before September 1994, they were important evidence available to Dr. Bragdon on the subject.

The first article concerns only cutaneous exposures to blood, and reported an approximately 50% reduction of cutaneous exposures by taking universal precautions. [637a-653a] This article does not suggest any significant reduction in the risk of HIV to dentists for several reasons. First, although "transmission of HIV infection after skin exposures also have been documented," Cardo & Bell, *Bloodborne Pathogen Transmissions in Health Care Workers*, Infectious Disease Clinics of North America, vol. 11, no. 2, p. 333 (June 1997), the risk for transmission after skin exposure was (and still is) thought to be far less than mucous membrane or percutaneous exposure. [557a]—"Risk of Infection Postexposure") Second, a 50% reduction in the risk of a fatal accident would hardly render

a risk insignificant if it was significant to begin with. Third, the article does not address the principal concerns of dentists, namely, mucous membrane and percutaneous exposures.

The second article, published in a March 1991 Journal of the American Medical Association ("JAMA"), considered parenteral (which includes percutaneous) exposures (the type of exposures most dangerous with respect to HIV). The second article states that "the implementation of UPs [i.e., universal precautions] should reduce the risk of occupational HIV infection by . . . 62%." [633a] Four months later, the authors implicitly admitted that the data upon which they relied was *not* statistically significant, and based on "the study data alone, the correct conclusion is that the implementation of UPs should *not* be expected to reduce either needle-stick injuries or the risk of HIV infection associated with them." [226a-229a] So much for what the CDC referred to in 1995 as the best and latest evidence concerning the efficacy of universal precautions for dentists in preventing the transmission of HIV.

Meanwhile, OSHA reported in its 1991 regulatory preamble that it could *not* quantify the benefits of universal precautions with respect to HIV, 56 Fed. Reg. at 64055, but the benefits of universal precautions with respect to hepatitis B virus ("HBV") were *insufficient* to provide the level of workplace safety mandated by law, *id.* at 64088. HBV is a bloodborne pathogen transmitted in the same way as HIV, except that the infectivity rates for HBV following percutaneous or mucous membrane exposures appear to be on the order of 25 to 100 times higher than HIV. *See* 56 Fed. Reg. at 64010 (reporting 7% to 30% infectivity rates following percutaneous exposure, compared to 0.3% to 0.5% for HIV). HBV mortality rates are far lower than HIV, on the order of 1%. *Id.* at 64009 (discussing HBV disease outcomes). Observed transmissions of HBV are therefore of some use in understanding the risk of transmission of HIV and the efficacy of universal precautions, keeping in mind that any given failure of universal precautions is perhaps 25 to 100 times more likely to result in HBV infection than HIV

infection.[13] Because HIV infection is fatal, whereas HBV infection usually is not, any observed failure of universal precautions with respect to HBV is significant for HIV.

A 1988 study released by Dr. Mark Kane of CDC and two others concluded that "the use of gloves and other protective devices does not appear to offer substantial protection against HBV exposure in oral surgeons." [400a] On the basis of the Kane study and other observed failures of universal precautions to prevent the transmission of HBV, and to prevent mucous membrane and percutaneous exposures generally, *see* 56 Fed. Reg. at 64033 (only 37% of mucous membrane and percutaneous exposures to patients' HIV-infected blood might have been prevented if CDC recommended infection control procedures had been followed), *see* [466a] ("[g]loves should reduce the incidence of contamination of hands, but they cannot

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[13] *See* [178a-179a] (Dr. Bell using observed HBV transmissions to test his HIV models); [573a] ¶ 9, [574a] ¶ 14 (Dr. Kuvin basing his opinions on HIV, in part, on observations of HBV); [308a] (Dr. Bragdon explaining HBV as model for HIV).

prevent penetrating injuries due to needles or other sharp instruments"), OSHA decided to require that employers, like Dr. Bragdon, offer HBV vaccine free of charge to their employees, 56 Fed. Reg. at 64088. There is, of course, no HIV vaccine, or as OSHA put it, "[m]oreover, the hepatitis B vaccine will not protect employees from other bloodborne pathogens such as HIV. Based on these data, OSHA has concluded that widespread administration of the hepatitis B vaccine will not eliminate significant risks," *id.* at 64037, or again, OSHA's mandate is "to reduce significant risks, which, in this case, would include risks remaining after administration of the hepatitis B vaccine," *id.* at 64038. Based on this analysis and other available data, objectively reasonable dentists could *not* have reasonably concluded that universal precautions would eliminate a direct threat of HIV.

Likewise, the then recent former Assistant Executive Director of Scientific Affairs for the American Dental Association (the "Association") conceded when pressed in March 1994 that there is *no scientific evidence*, but only

"intuition," that universal precautions are effective against HBV and HIV. [411a][14] Her admission is consistent with earlier studies suggesting no appreciable reduction of risk to dentists who adhere to universal precautions. *See* M.A. Noble, et al., *Hepatitis B and HIV Infections in Dental Professionals*, 57 J. Can. Dent. Assoc. 55, 55 (1991) ("use of gloves did not show a protective effect" and "[r]eliance on gloves for operator protection appears to be inadequate") [418a]. OSHA now expressly admits the obvious, namely, universal precautions and personal protective equipment are *not* adequate to protect the health care worker against needlestick injuries. [235a-236a]

Consequently, when the Guidelines state that their implementation "should reduce the risk of transmission of infectious diseases caused by bloodborne pathogens such as HBV and HIV" [529a], no objectively reasonable dentist in 1994 could have thought that "should" meant anything with

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[14] Similarly, then Secretary of the Association's Council on Dental Therapeutics admitted in May and July of 1990 there is no established scientific basis for using gloves in dentistry as a means to prevent the transmission of infectious diseases. [414a-417a]

respect to HIV than "might to a marginal extent." Objectively reasonable dentists in 1994 could have thought that "should" meant "probably will" with respect to HBV, but only because the Guidelines also recommended HBV vaccine for all dentists [530a-531a], not because of any demonstrated effect of gloves, masks, eye shields, or so forth.

B. The Guidelines Do Not Say Or Imply Universal Precautions Are The Best Way To Combat The Risk Of HIV Transmission. The Supreme Court said the Guidelines "set out CDC's recommendation that the universal precautions are the best way to combat the risk of HIV transmission." *Bragdon*, 118 S.Ct. at 2211. With all due respect to the Supreme Court, the Supreme Court is wrong. This is not what the Guidelines say or imply, especially when understood (as objectively reasonable dentists would have understood them) in the historical context of the lack of any scientific evidence of the efficacy of universal precautions to reduce the risk of HIV. *See supra* Part IV(A). The Guidelines expressly concede that events resulting in transmission of HIV infection in the dental setting "have not

been determined." [530a] Because no scientific evidence showed universal precautions reduced the risk of transmitting HIV, and because events resulting in the transmission of HIV infection in the dental setting "ha[d] not been determined," how could CDC be setting forth "the best way" to combat the risk of HIV? CDC understood this, but some members of the Supreme Court did not.[15] At most the Guidelines may be read as setting forth minimal acceptable, lowest common denominator precautions that "should" (but might not) reduce certain risks.

C. The Guidelines Do Not Say Or Imply That Universal Precautions Are The Maximum Desirable Precautions. The Guidelines expressly do *not* say universal

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[15] Because the justices who comprised the five member majority did not, by their own admission, attempt to review the record comprehensively, it is perhaps not surprising that they seem to assume the efficacy of universal precautions, i.e., wearing gloves, little eye shields, paper masks, etc. Common sense suggests that by wearing all that, dentists are less likely to get patients' blood on their skin, up their nose, and so forth. But viruses are very, very minute, and science has simply not shown that wearing all these things provides any statistically significant protection from the virus. Whether this is because of tiny holes in most gloves and masks, or the obvious fact that sharp instruments cut right through them, is not important. What is important is that no objectively reasonable dentist in 1994 could have concluded that universal precautions were effective, much less "the best" that can be done in specific settings.

precautions are the best or maximum desirable precautions in every setting. They say the opposite. They say they are the minimum precautions for every setting, but the "unique nature of most dental procedures, instrumentation and patient-care settings also may require specific [i.e., non-universal, thus additional] strategies directed to the prevention of transmission of pathogens." [528a-529a] Out of Dr. Bragdon's 8,000 patients, Abbott was the first one ever to inform him that she was HIV-positive. Consistent with the Guidelines (and expert advice to dentists that the "time, effort, and even dollars invested toward the pursuit of minimal risk is the most responsible option available to the health-care worker to provide both personal and patient confidence in establishing minimal disease risk" [243a], this should have given Dr. Bragdon the opportunity to take specific extra precautions, i.e., other "specific strategies directed to the prevention of transmission of pathogens." *See* [273a-299a]

[305a-307a] (describing such strategies).[16] *Cf. Lasser v. Rosa*, 654 N.Y.S.2d 822, 823 (App. Div. 1997) (dentist who offered HIV infected plaintiff referral to clinic which specialized in the treatment of HIV positive patients, and who explained that in his professional judgment these "clinics offered precautions and accommodations beyond the minimum standards prescribed by the [CDC]," was not liable for discrimination based on HIV status).

D. No Public Health Authority Available On Or Before September 1994 Supports The Position That There Is No Direct Threat. Apart from the Guidelines, Abbott cites no public health authority available on or before September 1994 to support her position that there is no direct threat to dentists --

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[16] Abbott estimates that the added cost to her would have been approximately $150. [255a] The American Dental Association reports that it costs dentists nearly $24,000 per practice per year to implement OSHA mandated universal precautions. [431a] Abbott was the only one of Dr. Bragdon's 8,000 patients ever to identify herself to him as HIV positive. From a purely economic perspective, Dr. Bragdon's recommended specific (i.e., non-universal) strategies for reducing the risk are *prima facie* modest. **Furthermore, Dr. Bragdon does not claim his strategies would have eliminated the significant risk. They might have done very little. But this is precisely consistent with the definition of direct threat under the statute: it is a significant risk "that cannot be eliminated." 42 U.S.C. § 12182(b)(3).**

none whatsoever. In the District Court, Abbott relied entirely on affidavits from Dr. Marianos, but as the Supreme Court stated, this Court's

> rejection of the District Court's reliance on the Marianos affidavits was a correct application of the principle that petitioner's actions must be evaluated in the light of the available, objective evidence. The record did not show that CDC had published the conclusion set out in the affidavits at the time petitioner refused to treat respondent [in his office].

Bragdon, 118 S. Ct. at 2211. The Marianos affidavits are therefore irrelevant and, in effect, inadmissible for purposes of determining whether Dr. Bragdon raises an issue for trial based on available, objective evidence in September 1994.

E. The American Dental Association's 1991 Policy Statement Has Little Or No Probative Value. The Supreme Court held that the American Dental Association "is *not* a public health authority," *Bragdon*, 118 S. Ct. at 2212 (emphasis added). Therefore, even if the American Dental Association's statements were admissible, they would carry no "special weight and authority," *id.* at 2211, especially where the issue is not one of

"dentists' ethical obligations" or "professional responsibility," *id.* at 2212. This is important to recognize at the outset because this Court has singled out for discussion a 1991 policy statement (the "Policy Statement") of the American Dental Association (the "Association") (App. 273). *See* Order of Court (Aug. 14, 1998) (requesting insights as "to the import and admissibility" of the Policy Statement).

The first important insight is the one already stated, namely, the Association is not a public health authority. Therefore, if the Policy Statement is admissible for the truth of the matters stated in it, it is entitled to at most the weight accorded opinions of regular, Rule 702 designated experts, such as Dr. Craven (for Abbott) or Dr. Kuvin (for Dr. Bragdon).

Second, there appears to be no hearsay exception by which the Policy Statement is admissible for the truth of the matter stated. In the District Court, both Abbott and the United States asserted the Policy Statement was a policy adopted by the Association, and they attached a copy to their briefs in the form

reproduced at [590a].[17] Dr. Bragdon did not dispute that the Policy Statement is statement of the Association. That is not an admission that the Policy Statement is admissible for the truth of the matter stated. Abbott did not designate the Association, or any official of it, as an expert witness; the Association is not a public office or agency to which the hearsay exception in Rule 803(8) might apply; nor does the Policy Statement seem like a "learned treatise" under Rule 803(18). The Policy Statement is therefore not admissible against Dr. Bragdon for the truth of the matters stated in it.

Third, even if the Policy Statement were admissible for the truth of the matters asserted, it would carry less weight than a normal expert opinion for several reasons. First, it is not the testimony of an expert. There is no accompanying vita or proof of credentials. There are no specific studies or data cited. There is no analysis. There is nothing but the barest of *ipse dixit*,

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[17] *See* Docket Doc. 59 (Pl.'s Mot. for S.J. filed 9/19/95), p. 9 (citing Exhibit L of Abbott's Record Appendix filed therewith.); Docket Doc. 53 (U.S. Statement of Material Fact filed 9/19/95), ¶ VIII.G (referencing attached U.S. Ex. No. 20).

which is usually of little or no probative value. *See Joiner*, 118 S. Ct. at 519.[18] Second, in this instance, there is evidence of the *unreliability* of the Association on such matters. We know that shortly before the Association adopted the Policy Statement (really, readopted it, since the identical language was previously adopted in 1988, *see* [525a], ¶ 17), the Association tried to convince OSHA there was no significant risk to dentists from HIV. OSHA reviewed the Association's evidence and expressly rejected it, explaining in December 1991 that the Association's "contention that the HIV risk in dentists is lower than that of the general population is confusing and is not supported by the data the [Association] cited in their posthearing comments." 56 Fed. Reg. at 64092. Unlike the Association, OSHA *is* a public agency for purposes of Rule 803(8). Unlike the Association, OSHA's published findings are entitled to the special weight of a public

[18] Abbott argued below that general dentists are not qualified as experts "to offer an expert opinion about any issue in this case." (Docket Doc. 61, p. 6, filed 9/19/95) The Policy Statement is *prima facie* a policy adopted by majority vote of a group of general dentists. Hence, if Abbott is correct, either the Association's statement is *prima facie* excludable as an opinion of mere general dentists, or it is the opinion of some unidentified third party or parties. If the latter, there is still another level of hearsay.

health authority. Thus the Association has a proven track record of offering confusing arguments not supported by the data. It would therefore be absurd for the Court, or an objectively reasonable dentist in 1994, to accord any significant probative value to the Association's 1991 Policy Statement.[19]

F. The Rule That Views Of Public Health Authorities Are Of Special Weight Is Not Applicable Against Dr. Bragdon In This Case. According to the Supreme Court, the views of public health authorities are of "special weight and authority." *Bragdon*, 118 S. Ct. at 2211. Their views are not, however, conclusive. *Id.* Although they establish a "prevailing medical consensus," *id.*, such a consensus may be refuted by citing "a credible scientific basis for deviating from the accepted

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[19] In addition, the contemporaneous opinion of the American Association of Orthodontists in 1991 was that in health care settings "scientific evidence indicates that HIV disease poses a direct threat and significant risk" and "much uncertainty surrounds what precautions, including barrier techniques, if any, can prevent the transmission of HIV disease." Comment No. 863 at 5 (April 23, 1991) in ADA Comment Book #1 for Public Inspection, Justice Department, disability rights division, 4th floor, 1425 New York Ave. N.W., Washington, D.C. As a public document in the custody of the Justice Department, the Court may take judicial notice of it under Rule 201. Relevant portions of the letter are reproduced at [435a-438a]. This shows disagreement among professional dental associations on the direct threat issue.

norm," *id.*

In this case, the Court does not need to reach, and should not reach the issue of how "special" is the "special weight and authority" to be accorded the views of public health authorities. This is because, as the previous discussion shows, no such views in support of Abbott were available in September 1994, and therefore there is no "prevailing medical consensus," *id.*, for Dr. Bragdon to refute. Instead, the Court needs to approach the direct threat issue as it would any other summary judgment dispute. The Court must not accord Abbott's authorities any "special weight," it must demand of Abbott as much scientific rigor as it demands of Dr. Bragdon, it must eschew fact finding, it must not make credibility determinations, it must believe the evidence of Dr. Bragdon, and it must draw all reasonable inferences in his favor. *Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 255 (1986). When it does, it will agree with four members of the Supreme Court who think "it is clear" that Dr. Bragdon presented enough evidence to avoid summary

judgment. *Id.* at 2217 (Rehnquist, C.J., dissenting, joined as to Part II by Scalia, Thomas, and O'Connor, JJ.).

V. THERE IS NO SCIENTIFIC VALIDITY TO ABBOTT'S ARGUMENT THAT BECAUSE THERE HAS NEVER BEEN A "DOCUMENTED" CASE OF HIV TRANSMISSION TO A DENTIST, THERE IS NO DIRECT THREAT.

Abbott has repeatedly argued there is no significant risk to dentists because there has never been a documented case of HIV transmission from infected patient to dentist in over 1,000,000,000 procedures performed in the past 14 years. Her implied premise is that with all of those opportunities for transmission, if the risk were at all significant, there would have been at least one documented case. Her argument is logically and scientifically absurd for the following reasons, especially when looks at the issue from the standpoint of an objectively reasonable person in September 1994.

A. 42 Documented Transmissions To Health Care Workers. As of September 1994, CDC reported 42 documented occupational transmissions of HIV to health care workers, but

none to dentists. [224a][20] As of that date, no scientific evidence supported the conclusion that something other than chance was the reason there was no documented transmission to a dentist. In other words, it would not have been objectively reasonable for a dentist to infer from anything said in the CDC's June 1994 article at [549a-566a], or from any other source available at the time, that the risk to dentists was significantly less than other categories listed on Table 11 at [224a]. Abbott's only conjecture in this regard has been that dentists use smaller needles and there is usually less blood on dental devices. Yet objective sources at the time recognized (as they do today) that superficial scratches or needles with no visible blood can transmit HIV. [188a, 201a, 380a, 386a,394a-395a] In addition, there is often visible blood on sharp dental devices. [340a]

Furthermore, Abbott's theory fails to explain why there are 6 documented cases of transmission to non-surgical

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[20] CDC's June 1993 figures are found at [550a] in an article from CDC published in June 1994. CDC's June 1994 figures are found at [224a], in HIV/AIDS Surveillance Report, vol. 6, no. 1, p. 15, tbl. 11 (Mid-year ed. June 1994), cited by the Supreme Court, *Bragdon*, 118 S.Ct. at 2212.

physicians, but *none* to surgical physicians. [224a] On her theory, surgical physicians should be at greater risk. And what about emergency medical technicians/paramedics? Blood and sharp objects are often all over the place where they work, but there is no documented case of transmission to one of them. (*Id.*) Does that mean surgical physicians and paramedics are infinitely less at risk than non-surgical physicians, for whom there are 6 documented cases? Of course not. *Cf. Abbott*, 107 F.3d at 947 (health-care workers in "emergency rooms may be exposed to much larger needles and more copious quantities of blood"). Yet this is the unscientific reasoning Abbott relies on when she tries to read statistical or empirical significance into the lack of a documented cases for dentists in Table 11.

CDC certainly does not suggest in the June 1994 article (or June 1994 surveillance report) there is any such signifieance. [549a-566a, 224a] On the contrary, CDC provides a sound scientific explanation for what might otherwise look like numerical anomalies in the table, namely, CDC's

> passive surveillance system for occupational transmission most likely does

> not reflect the full extent of occupational transmission; such cases can be difficult to document, particularly if exposures are not reported at the time of occurrence. In the past, it has been estimated that only 10% to 60% of percutaneous injuries are reported. [549a]

That is why there is little or no objective significance to the lack of a documented transmission to a surgical physician, emergency medical technician, or dentist in 1994. That is why objectively reasonable dentists in 1994 could not conclude from the lack of a documented transmission that there is no direct threat. Four members of the Supreme Court understood this and summed it up by saying "the fact that the health care workers were not dentists is no more valid a basis for distinguishing these [documented] transmissions of HIV than the fact that the health care workers did not practice in Maine." *Bragdon*, 118 S. Ct. at 2217 (rejecting this Court's previous suggestion that the 42 documented transmissions are irrelevant). The 42 documented transmissions illustrate the invalidity of Abbott's argument and by themselves and in combination with other evidence raise a genuine issue of direct threat.

B. 88 Possible Transmissions To Health Care Workers. CDC also reported 88 possible occupational transmissions of HIV to health care workers as of September 1994. [224a] This included 7 possible transmissions to dental workers (including dentists). (*Id.*) This means that an objectively reasonable dentist, relying on the best information in September 1994, would have known the following: CDC reported 88 instances of HIV-infected health care workers, including 7 dental workers; these HIV-infected workers reported "percutaneous or mucocutaneous occupational exposures" to HIV, they were "investigated and are without identifiable behavioral or transfusion risks" for acquiring HIV, but "HIV seroconversion specifically resulting from an occupational exposure was not documented." This means that the workers did not take a blood test at the time of exposure to establish HIV-negativity at the time, but were tested some time later and that

test was positive. (*Id.*)[21]

Abbott's argument with respect to the 7 dental workers (and presumably the 81 other cases reported as "possible") is yes, they have HIV, they reported percutaneous or mucocutaneous exposure to HIV on the job, and investigation revealed no non-occupational risk factors; nonetheless, an "objectively reasonable" dentist reading this should have concluded that everyone was infected while they carried on illicit sexual affairs or intravenous drug use hidden from CDC investigators, or some such thing, and therefore the risk to dentists is insignificant. [616a] ¶ 5) If this were so, one wonders why CDC even bothered to report such cases, if they have no probative value.

The opposite is true. These cases have probative value in medical science and formulation of public health policy. For example, in the Klein study discussed by OSHA, 56 Fed. Reg. at

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[21] Standard blood tests for HIV detect the presence of HIV-antibodies that do not appear for several weeks after the exposure. *Bragdon*, 118 S. Ct. at 2203-04. A blood tests done at the time of exposure can therefore establish a baseline seronegativity with "documented" seroconversion later on if and when antibodies develop.

64092, there is also what the CDC would classify as a "possible" transmission to a dentist, namely, an HIV-infected dentist with reported occupational percutaneous exposure to HIV, no documented seroconversion from a specific exposure incident, and no behavioral risk factors. [353a-356a] What do the team of scientists who conducted the Klein study conclude? They conclude that the evidence "suggests that occupational exposure was the likely mode of acquisition of his HIV infection." [355a] Is this "scientific certainty"? No. Is it scientific? Yes. And what does OSHA conclude? It concludes that the Klein study suggests there is a significant risk to dentists, where "significant" means approximately one likely fatality per thousand workers exposed. 56 Fed. Reg. at 64092. Now, if this form of inference is good enough for the 7 authors of the Klein study, good enough for the *New England Journal of Medicine* which published the study, and good enough for OSHA, how in justice may it not be good enough for a reasonable dentist three years later reading CDC's June 1994 surveillance report? The conclusion that 7 possible transmissions of HIV to dental workers raises a genuine

issue for trial is "derived by the scientific method," *Daubert*, 509 U.S. at 590, appropriate for medicine, medical science, and public health policy. It would be unscientific and contrary to law for the Court to dismiss these 7 reported cases of dental workers with HIV as having no probative value.

With all due respect to the Supreme Court, the majority's discussion of the 7 possible transmissions and whether people knew in 1994 "whether HIV infection had resulted," *Bragdon*, 118 S. Ct. at 2212, makes no sense because it is based on flat-out confusion. Specifically, the Supreme Court somehow thought that when CDC reported "possible" transmissions, that meant instances in which CDC was uncertain whether the workers were actually infected with HIV. The Court apparently made this mistake because it confused the 19 dental workers discussed in Gooch et al. [335a], cited at *Bragdon*, 118 S. Ct. at 2212, with the reported possible transmissions to dental workers set forth in CDC, HIV/AIDS Surveillance Report, vol. 6, no. 1, tbl. 11 [224a], also cited at *Bragdon*, 118 S. Ct. at 2212. These are different studies/reports involving different dental workers, as

Dr. Marianos explains at [616a] ¶ 5. The 19 dentists discussed in the Gooch article are among over 2000 health care workers enrolled in an on-going study that tried to fix more precisely the rate of HIV infection after percutaneous exposures to HIV. The study arrived at a figure of approximately 0.36%. [318a] A worker does not get into this study unless he has had a percutaneous exposure *and* a test at the time of exposure to establish he was HIV-negative at the time. [319a-320a] In theory, he is then supposed to be tested several weeks and several months later to see if he has seroconverted, i.e., become HIV-positive. (*Id.*) But as Gooch reports, among the 19 dentists in the study, 7 did not come back for proper follow up to determine whether they did or did not seroconvert (of the 12 who did come back for proper testing, none seroconverted). [339a] The Supreme Court apparently confused the 7 dentists who did not come back for proper testing with the 7 reported possible transmissions to dental workers. But as already explained, the 7 dental workers (and 81 other health care workers) whom CDC reported as "possible" transmissions are all people who were

already infected with HIV, who contacted CDC, who reported occupational HIV exposures, and for whom CDC conducted follow up investigations in order to identify possible non-occupational risk factors. The latter is the "passive surveillance system" whose limitations are described at [549a]. Somehow the Supreme Court confused the passive surveillance system with the Gooch article, and wondered whether Dr. Bragdon could have known in September 1994 (since the Gooch article appeared in September 1995) that none of the 7 "possible" cases resulted in HIV infection, *Bragdon*, 118 S. Ct. at 2212, which makes no sense since everyone knew in September 1994 that all

7 dental workers were already infected.[22]

C. The Number Of CDC Reported Possible Transmissions To Dental Workers Accords With Dr. Kuvin's Calculation Of The Risk Per Dental Procedure. According to CDC, there were probably between 650,000 to 900,000 persons in the United States infected with HIV at end of 1993. [446a-447a] No one knows how often these persons went to the dentist and had an invasive procedure (e.g., cavity filled or tooth extraction). CDC estimates 25% are injecting drug users. [441a] Common sense suggests many injecting drug users do not go to dentists for regular check-ups, as is true for another overlapping group at high risk for HIV, i.e., prostitutes. Also, at times prior

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[22] No expert thinks there is statistical significance in the fact that among the 12 dentists discussed by Gooch, none seroconverted. With an observed seroconversion rate of only 3 to 4 conversions per 1000, no seroconversions among the 12 dental workers, with 7 others not reporting, is not statistically significant. In addition, 6 of the 19 exposures were instances of visible blood on the device [340a], which CDC reported three months later is a factor that significantly increases the risk of HIV infection following percutaneous injury [665a]. In her article, Gooch tries to downplay the significance of so many instances of visible blood on devices with the lawyer-like statement "overall, these dental workers may have been exposed to smaller rather than larger quantities of blood." One must suppose that by "overall" she means 13 of 19 instances. Yet given the importance of any visible blood on the device, the truly important finding is that dentists are often at significantly above average risk for infection following percutaneous injuries.

to 1994, there would have been significantly fewer infected persons. In addition, many persons (especially young adults) who regularly visit a dentist often go five or ten years needing nothing but routine cleanings. Nevertheless, assume that between 1982 and 1994, there were 775,000 persons infected with HIV, and that every one of them had 3 invasive procedures performed on them by an uninfected dentist with no identifiable non-occupational risk factor for HIV. The latter assumption is required because if a dentist is already infected, or has other risk factors, CDC will not consider it an occupational transmission. Three invasive procedures per HIV-infected patient times 775,000 HIV-infected patients is 2,325,000 invasive procedures performed on HIV-infected patients prior to 1994. Assuming 2 percutaneous injuries per 100 procedures (a higher rate than 1.5 per 100 injections used by Dr. Kuvin), and Dr. Kuvin's contaminated instrument rate of 1 in 3, there would have been 2,325,000 x 2/100 x 1/3, or 15,500 percutaneous injuries in which uninfected dentists without identifiable non-occupational risk factors were percutaneously exposed to HIV. Using a 0.3%

infectivity rate (as did Dr. Kuvin), there would have been about 15,500 x 3/1000, or 46 ***potentially*** CDC reportable occupational transmissions to dentists between 1982 and 1994. If instead of using 2 percutaneous injuries per 100 procedures, one uses Dr. Bell's estimate for dentists of 0.4 per 100 [180a], the result is only 9 ***potentially*** reportable occupational transmissions to dentists. Of these estimated 9 to 46 potentially reportable transmissions, how many would likely actually have been reported by dental workers to the CDC, and then reported by the CDC by September 1994?

Because it takes on average 7 to 11 years for AIDS-like symptoms to develop, *see Bragdon*, 118 S.Ct. at 2204, many of the 9 to 46 dental workers would not yet know in 1994 they had anything to report. As Dr. Bell explained in 1991 with respect to the analogous problem of identifying patients who had unknowingly contracted HIV, "[t]o date only a small fraction of this theoretical patient pool would have developed AIDS and been detected through AIDS surveillance." [180a] Assuming, as does CDC, that 1 in 3 persons infected with HIV in the United

States does not know it [450a], only about 6 to 30 of the 9 to 46 dental workers would even know they had HIV. Furthermore, dentists among the 6 to 30 who contracted HIV while working years earlier on patients who did not know they had HIV, or who did not tell their dentists, would have nothing reportable by CDC, since these dentists can not trace their HIV-infections back to a known exposure to HIV-infected blood. These are reasons why CDC said its passive surveillance system "most likely does not reflect the full extent of occupational transmission," with an estimated 40% to 90% of percutaneous injuries going unreported [549a]. These are reasons why, if Dr. Kuvin's calculation of the risk to dentists per injection given to an HIV-infected patient is reasonably accurate, one would expect considerably fewer than between 6 to 30 CDC reported documented or possible transmissions to dentists by 1994. As it happens, there were 7 reported possible transmissions to dentists at that time. (Add. 43) This is consistent with and tends to support Dr. Kuvin's

calculation of the risk.[23]

VI. OTHER EVIDENCE, BOTH BY ITSELF AND AS CUMULATIVE WITH EVIDENCE ALREADY DISCUSSED, RAISES A GENUINE ISSUE FOR TRIAL.

A. OSHA Sets A Baseline Of Significant Risk Not Eliminated By Universal Precautions.

In 1991, OSHA promulgated its Final Standard for Occupational Exposure to Bloodborne Pathogens, 56 Fed. Reg. 64004 (Dec. 6, 1991). The standard applies to dental workers. *E.g., id.* at 64092. OSHA concluded that dental workers are "at risk" of contracting HIV, and that the rate of HIV infection is low, but "not insignificant." *Id.* at 64032. Logically, "not insignificant" means significant, unless an infection rate can be neither significant nor insignificant, which makes no sense without some sort of explanation. Moreover, OSHA *specifically rejected* the

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[23] Abbott's analysis also begins with the wrong number of procedures, i.e., "over one billion." One billion is her estimate of the number of procedures performed on all patients, over 99% of whom do *not* have HIV. [451a] As Dr. Kuvin explained, it is fallacious to imply, as does Abbott, that the risk of transmission of HIV from patient to dentist is negligible because patients without HIV have not transmitted HIV to their dentists. [607a] ¶ 19(a)) Instead, one must begin with an estimate of the number of procedures performed on HIV-infected persons by uninfected dentists, as does Dr. Bragdon's analysis in this Part V(C).

American Dental Association's contention that OSHA overestimated the occupational HIV risk for dentists. *Id.* at 64092. OSHA explained that dental workers "are continually exposed to blood and bloody saliva during almost all dental procedures," *id.*, and that empirical findings tend to suggest an incidence of occupationally acquired HIV among dentists that is not "minimal" or "insignificant," but "significant" as defined by Supreme Court precedent, *id.* In fact, OSHA deemed the risk so significant that for the first time in history every dental worker was required to treat every patient as though the patient were infected with HIV, at an annual cost of approximately $24,000 per dental practice, even though over 99% of the population was not infected. [431a, 440a]. This was a crystal clear message to dentists from public health authorities that there was a direct

threat from HIV.[24] Likewise, CDC earlier concluded that HIV was such a serious threat in the dental setting that "blood, *saliva*, and gingival fluid from **all** patients should be considered infective" (bold underline in original, other emphasis added). [460a] CDC recommended such extraordinary measures for dentistry in part due to the same "possible" occupational transmissions to dentists which Abbott now insists, by a double standard, are of no probative value. ([466a] (explaining rationale for "special precautions" in dentistry)) The burden should therefore be on Abbott and the CDC to explain why a droplet of saliva from a child with no HIV risk factors requires universal precautions if no objectively reasonable dentist could think there was a direct threat of HIV.

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[24] OSHA's bloodborne pathogen standard was also ostensibly promulgated due to the threat of HBV. However, there is an HBV vaccine, *see* 56 Fed. Reg. at 64029, and even if every dental worker in a dental practice has taken the vaccine and had blood tests to prove immunization was effective (by measurement of hepatitis B surface antibody), OSHA still requires all the same precautions due to the HIV threat alone. It was also commonly understood that the driving force behind the regulation was the new threat of HIV (especially the July 1990 CDC report of the transmission of HIV from a dentist to patients), and not HBV, since "by the mid-1970's [HBV] was known to be at a much higher prevalence in dentists than in the general population." [332a]

Similarly, how can the threat to dental workers in 1994 have been deemed insignificant for summary judgment purposes when under the OSHA standard, 29 CFR 1910.1030(f)(3)(ii)-(vi), every time there is an "exposure incident" with *any* patient the dental worker (and patient, if the patient consents) must be sent to a licensed physician for post-exposure HIV counseling and possible PEP? Guidelines state that the post-exposure HIV evaluation by a physician and the commencement of PEP, if appropriate, should occur as quickly as possible, preferably within 1-2 hours. [477a][25] This is another reason why Dr. Bragdon wanted to fill Abbott's cavity in a hospital, so that if he or his assistant were accidentally stuck (or by today's standard, superficially scratched), he or his assistant could receive immediate attention, thereby most fully complying with OSHA standards and CDC recommendations. *See* [208a] ("PEP should be initiated as soon as possible"). Abbott's supporters are

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[25] OSHA and the American Dental Association recently published a guide for dentists concerning these requirements, *see* [473a], but since the guide explains OSHA requirements already in place for dentists like Dr. Bragdon in September 1994, it is a useful summary. In particular, CDC had already indicated in 1990 that PEP is likely most effective if commenced as soon as possible after the exposure. [471a]

therefore ignorant when they accuse Dr. Bragdon of unprofessional behavior. In any event, CDC and OSHA sent these clear messages to dentists as of September 1994 that they and their staff faced significant risks calling for unprecedented precautions and immediate medical interventions.

OSHA and CDC's clear messages of a direct threat to dentists set the "initial baseline degree of danger of treatment in the absence of the universal precautions." *Abbott*, 107 F.3d at 948 (implying that Dr. Bragdon had no such baseline from which to assess the impact of universal precautions). The question then becomes what was the scientific evidence available to Dr. Bragdon as to the efficacy of universal precautions in reducing the threat of HIV to dental workers? The answer, as already explained, is that an objectively reasonable dentist in 1994 could not have concluded that universal precautions reduced the threat of HIV. *See supra* Part IV(A). Based on objectively reasonable judgment and then-current medical knowledge, Dr. Bragdon could have therefore concluded, and did conclude, that performing an invasive procedure on Abbott

would have posed a direct threat pursuant to 28 C.F.R. § 36.208(c).

B. FDA Recommendations. In 1992, the Food and Drug Administration (FDA) issued recommendations for the prevention of HIV transmission. The FDA recommended a twelve month deferral period for all potential blood donors "who have had contact with blood and body fluids through percutaneous inoculation (such as injury or accidental needlestick) or through contact with an open wound, non-intact skin, or mucous membrane during the preceding 12 months." [601a] This means that whenever a dentist has contact with *any* patient's blood by accidental needlestick or other sharps injury, the FDA, on account of concern for the transmission of HIV, recommends that the dentist not be permitted to donate blood for twelve months, to ensure that blood tests will detect HIV-antibodies if he is infected. It is therefore reasonable to infer that the FDA, like OSHA, thinks there is a significant risk to dentists of contracting HIV when they are exposed to the blood of their patients.

C. HIV Transmissions to Dental Patients. CDC reported that a Florida dentist, despite using barrier precautions, transmitted HIV to five of his patients, between 1987 and 1989. [357a] The routine procedures in question included restorative fillings, prophylaxes, and simple extractions. The CDC explained that

> [a]ll five patients received multiple injections of local anesthetic, and a sharps injury during anesthetic administration could have resulted in contamination of the syringe apparatus with the dentist's blood. . . . A sharps injury could also result in direct contact of the dentist's blood with the patient's inflamed or nonintact oral tissues during the invasive procedures. Although the dentist began to routinely wear gloves in 1987, gloves do not prevent most injuries caused by sharp instruments.

[372a] The CDC updated this study in 1993, and concluded that a sixth patient was also infected with HIV while receiving care from this dentist. Unlike the other patients, "this patient had neither dental extractions nor root canal therapy." [378a] Other than X-rays and a cleaning, there was only a single surface restorative filling, like the "routine procedure" at issue in this

case. Experts concede the risk of transmission of HIV from patient to dentist is greater than the risk of transmission of HIV from dentist to patient. *E.g.* [191a]. Given (i) confirmed transmissions of HIV from dentist to patients while filling a simple cavity and other "routine procedures," (ii) courts are in agreement there is a direct threat to patients from HIV infected health care workers, including dentists,[26] (iii) the issue under statute is not ethics or professional responsibilities, (iv) the risk to dentists is greater than the risk to patients, and (v) dentists cannot control unexpected patient movements that cause percutaneous injuries [577a] ¶ 7; [618a-620a], Dr. Bragdon has at least created a genuine issue of direct threat.

D. Risk of Aerosol Transmission and Burden of Proof Generally. Dr. Bragdon introduced an affidavit from Leon Kelleher, D.D.S. [666a-669a] Dr. Kelleher directs a group of 17 dentists who treat over 100 HIV-positive patients. [667a]

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[26] *E.g., Bradley v. University of Tex. M.D. Anderson Cancer Ctr.*, 3 F.3d 922 (5th Cir. 1993), *cert. denied*, 114 S. Ct. 1071 (1994); *Mauro v. Borgess Medical Ctr.*, 886 F. Supp. 1349 (W.D. Mich. 1995), *aff'd* 137 F.3d 398 (6th Cir. 1998); *Doe v. Washington Univ.*, 780 F. Supp. 628 (E.D. Mo. 1991).

He testified their "standard operating procedure is to avoid the use of aerosol producing instrumentation [i.e., the dentist's high speed drill] in known HIV+ cases." [668a] According to Dr. Kelleher, "[p]atients on whom the use of the high speed handpiece [i.e., the drill] is unavoidable are done with high volume evacuation under laminar flow conditions with hood or full face protection and remote air source for the operator." (*Id.*) A similar type of breathing apparatus is described by Drs. Weber and Wulc in a 1992 article in Annals of Plastic Surgery. [479a] Dr. Kelleher's group adopted these respirator protocols due to a 1991 study conducted by Drs. Johnson and Robinson at Stanford University School of Medicine. [667a] Drs. Johnson and Robinson reported that infectious HIV can be isolated from aerosols generated in the laboratory from HIV infected blood. They concluded that

> In the absence of definitive studies proving that aerosols in clinical settings pose no risk of HIV transmission to personnel, . . . dentists . . . should avoid techniques which generate such aerosols . . . with patients known to be infected with HIV. Where such procedures are unavoidable, personnel should consider

> using high efficiency filtration masks and systems for containing aerosols. [660a]

When Dr. Bragdon fills a cavity such as Abbott's, the drilling process creates an aerosol spray of tiny droplets of blood and bloody saliva that drift up into his face. [577a] ¶ 10) One 1992 textbook for dental technicians describes this spray as "the equivalent of having someone sneeze in your face twice per minute—at a distance of 1 foot." [522a] CDC estimated in 1994 (as they do today) that the infectivity rate following mucous membrane exposure to HIV (e.g., exposure to tissues up the nose or in the mouth) is 0.09%, which is about 1 in a 1000. [557a] If Dr. Bragdon had filled Abbott's cavity using only a traditional surgical mask, airborne particles could have flowed around the imperfect seals creating a situation in which, as Drs. Weber and Wulc put it, "danger exists." [482a] To eliminate this danger, Dr. Bragdon wanted to defer treatment to a hospital setting where he believed such breathing apparatus would be available, or he could rent them, and there would be space for him and his assistant to use them. [284a]

The Supreme Court characterizes Dr. Kuvin as conceding that "no evidence suggested the spray could transmit HIV," and that his opinion on "airborne risk was based on the absence of contrary evidence." *Bragdon*, 118 S.Ct. at 2212. The Supreme Court overstates the matter in light of the context at 586a-589a] and Dr. Kuvin's testimony at App. 362. Contrary to what the Supreme Court says, Dr. Kuvin's testimony at [602a-603a] does have "a traceable, analytical basis in objective fact," *Bragdon*, 118 S.Ct. at 2212, namely, the results of the Johnson & Robinson study, the published findings of Chitwood and Ho showing that minute amounts of blood may transmit HIV [380, 395], and the approximately 1 in a 1000 risk of acquiring HIV following mucous membrane exposure. [557a] The problem that the Supreme Court sees is that the science in this area is especially inconclusive because unlike documented transmissions of HIV following splashes of blood to mucous membranes (e.g., 56 Fed. Reg. at 64018 (case 17)), there are none following mucous membrane exposures to aerosols. So what is the law to do? The answer depends on two things: first,

who has the burden of proof, and second, regardless of that, does Title III of the ADA permit dentists to act in clinical settings as reasonably prudent persons would act based on recommendations in published laboratory findings.

1. Between unfounded fear and scientific proof there is sound practical reason, and Congress did not intend to impose civil rights liability on those who act in conformity with sound practical reason in the absence of scientific proof. Consider what should happen if a person comes up to someone and offers to spray in his face an aerosol mist of bloody, HIV-infected saliva, and says he can either wear an ordinary surgical mask or one of Dr. Kelleher's respirators. What is the smart thing to do scientifically? Medically? Politically? Ethically? Legally? Is it unfounded fear to wear the respirator, so that the government should prohibit the wearing of one, or make dentists spend thousands of dollars remodeling their offices for one patient instead of going down the street to a hospital if they want to wear one? Or is it sound practical reason to go down the street and wear one? After all, Drs. Johnson & Robinson did not

just conclude that more work is required to see if a risk exists in a clinical setting [654a], as the Supreme Court implies; they also concluded that in the absence of studies showing there is no risk, "dentists . . . should avoid techniques which generate such aerosols," and if it is "unavoidable," they "should consider using [respirators]." [660a] Under these circumstances, Congress could not have intended to impose civil rights liability on Dr. Bragdon for wanting to wear a respirator simply because that meant offering to do a procedure in a hospital.

2. The Burden Of Proof Is On Abbott To Show There Is No Direct Threat. In four separate subsections, 42 U.S.C. § 12182 expressly puts the burden of proof on defendants.[27] Subsection (b)(3), the "direct threat" provision, does not. Nor does it characterize "direct threat" as a "defense." This means

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[27] *See* 42 U.S.C. § 12182(b)(2)(A)(ii) (defendant must prove modifications alter nature of service), (b)(2)(A)(iii) (defendant must prove undue burden), (b)(2)(A)(v) (defendant must prove barrier removal not readily achievable), (b)(2)C)(ii) (defendant must prove service equivalency).

either Congress intended for the burden to be on the plaintiff, as it normally would be, *see* 21 Wright & Graham, *Federal Practice and Procedure: Evidence* § 5122 n.12 (1977), or the statute does not assign the burden. If the statute does not assign the burden, the assignment is "'a question of policy and fairness based on experience,'" *Mashpee Tribe v. New Seabury Corp.*, 592 F.2d 575, 589 n.13 (1st Cir. 1979), including which side has greater control of evidence, and generally, on whom the burden would "sit lightest," *id.* n. 15; *cf. E.E.O.C. v. Amego, Inc.*, 110 F.3d 135, 144 (1st Cir. 1997) (burden on Title I employee to prove no direct threat when job implicates safety of others, and reserving burden of proof issue under Title III).

When evidence is inconclusive but only time or money is at stake, as in typical claims of "undue burden," it makes sense to put the burden on defendants, which is what the statute does, 42 U.S.C. § 12182(b)(2)(A)(iii). But when evidence is inconclusive and lives are at stake, as in a typical claim of direct threat, the burden should be on the plaintiff. *Cf. Jairath v. Dyer*, 972 F. Supp. 1461, 1470 (N.D. Geo. 1997) ("The Court finds no

reason to penalize a doctor for exercising caution in his medical judgments. Use of caution . . . is to be distinguished from situations in which doctors blatantly discriminate against patients"), *vacated on other grounds*, ___ F.3d ___ (11th Cir. 1998). Furthermore, a person with a disability is usually much more familiar with the disability's nuances than a public accommodation, having lived with it and having had the advice and counsel of specialists and supporting organizations. The average public accommodation is far less likely to understand and appreciate the medical facts and to have ready access to specialists in the field. So too in this case, where Abbott came to Dr. Bragdon at the urging of the Eastern Maine AIDS Network [485a-487a], and brought with her in litigation the resources of the State of Maine and the United States, including Dr. Marianos of CDC, as well as two experts of her own, all of whom swear that Dr. Bragdon's professional judgment, as a general dentist, cannot be trusted. Then Abbott brought with her on appeal several more public health organizations [493a], the American Medical Association [499a], and now the American Dental

Association *who refuses to share information about its 1991 Policy Statement with Dr. Bragdon* [506a-513a]. The burden would sit very, very lightly, indeed, on all of them to prove there is no direct threat, or that universal precautions are effective, if that were true. The burden should be on them to prove why CDC recommends PEP for superficial scratches if, as they claim, the risk to Dr. Bragdon from scratches is trivial. The burden should be on them to prove why, as they claim, visible blood on dental devices is not enough blood for dentists to worry about. The burden should be on them to prove why CDC bothers to report possible transmissions to dentists if, as they claim, possible transmissions have no probative value. The burden should be on them to prove, as they claim, Dr. Kuvin's risk analysis is wrong. The burden should be on them to present a meaningful risk analysis. In short, the burden should be on them to prove there is no direct threat. If they cannot, Dr. Bragdon should not be held liable for offering to treat Abbott in a hospital and refusing to perform the procedure in his office.

CONCLUSION

Abbott has not met her summary judgment burden of presenting evidence that a reasonable dentist could have concluded there was no direct threat based on medical or other objective evidence on record as of September 1994. Summary judgment should therefore be awarded to Dr. Bragdon. In the alternative, Dr. Bell's and Dr. Kuvin's calculations; Dr. Kuvin's direct affidavit testimony unchallenged in the District Court under *Daubert* or Rule 702; the conceded greater risk to dentists and other health care workers than to their patients; the reported possible occupational HIV transmissions to dental workers; the confirmed transmissions of HIV to dental patients; the documented and possible HIV transmissions from other patients to health care workers; the levels of risk deemed significant by the Supreme Court, the common law, OSHA, reasonable persons, and public health officials in the past; reports and studies of sharps injuries among dentists and Dr. Bragdon's testimony as to his rate of sharps injury; reported rates of HIV infection following percutaneous injuries; the Klein study; CDC PEP recommendations after superficial scratches and the fact

that superficial scratches have resulted in HIV transmission; the lack of scientific evidence that universal precautions prevent the transmission of HIV; OSHA findings and FDA recommendations; and every other piece of evidence discussed herein by itself and cumulative with the others, raises a genuine issue for trial as to whether, from the standpoint of Dr. Bragdon, was there a direct threat based on medical or other objective evidence.

Dated: September 28, 1998

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CERTIFICATE OF SERVICE

I, Brent A. Singer, Esq., certify that on September 28, 1998, I served by first class mail, postage prepaid, two copies of this paper Brief and one copy of the same on a computer disk in Word Perfect format on counsel for each party separately represented, specifically, two copies on Bennett H. Klein, Esq., G.L.A.D., 294 Washington Street, Suite 740, Boston, MA, 02108, and two copies on John E. Carnes, Esq., Maine Human Rights Commission, State House Station 51, Augusta, ME 04333.

/s/ Brent A. Singer, Esq.
Brent A. Singer, Esq.

CDC HIV/AIDS
PREVENTION

Open Meeting on the Risks of Transmission of Bloodborne Pathogens to Patients During Invasive Procedures

February 21-22, 1991
Hyatt Regency Atlanta
Atlanta, Georgia

HIV/ODD(HIV)/3-91/992

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control

CDC
Centers for Disease Control

[Add.1]

OPEN MEETING ON THE RISKS OF TRANSMISSION OF BLOODBORNE PATHOGENS TO PATIENTS DURING INVASIVE PROCEDURES

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Atlanta, Georgia

Transcribed by Barbara Hilger, RPR

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[Add. 2]

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the dentist and we are concentrating on the hygienists, often the hygienists would process their own instruments in the sense they would take them to the central sterilization unit and they would be washed and scrubbed and placed into the disinfectant solution, if that was indeed what would happen at the time. If there was time available, they would be autoclaved. But the hygienists for the most part processed their own instruments.

DR. CURRAN: I'd like to thank our speakers and resource panel and those that commented from the floor for the first session.

Our next speaker is Dr. David Bell, Chief of the AIDS Activity of the Hospital Infections Program at the Centers for Disease Control, and he'll be discussing the draft risk assessment model that you received.

DR. BELL: Thank you and good morning. In my talk today I will discuss some of the major points in the CDC draft risk assessment document. I will concentrate primarily on HIV transmission. Can I have the first slide please.

The risk of HIV transmission from an infected dentist or surgeon to a patient during an invasive procedure has not been extensively studied.

[Add. 3]

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As discussed by Drs. Jaffe and Myers, investigation of patients of a dentist with AIDS in Florida strongly suggested that HIV

was transmitted to three or 0.4 percent of 779 patients who have been evaluated to date.

Recently a dental student at the Medical College of Georgia was diagnosed with HIV infection. Most procedures performed by the student appear to have been relatively noninvasive. In collaboration with the Georgia Department of Human Resources, the college offered counseling and testing to the 163 patients who had been treated by this student. Preliminary results indicate that at least 146 patients have been tested and all are negative.

Three studies have been reported in which HIV testing was offered to patients of surgeons with AIDS. In the first two studies, 75 and 62 patients respectively of a general surgeon and a surgical resident with AIDS were tested. All were negative, although in the second study most patients were tested less than 90 days after surgery. In the third study HIV testing was offered to patients of a general surgeon in Nashville who had been operated on within seven years before the surgeon's diagnosis of AIDS. Of 1,340 patients who were contacted, 616 were tested.

[Add. 4]

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One patient, a known intravenous drug user, was HIV positive and may already have been infected at surgery.

The results of this large and well-conducted study illustrate some of the difficulties in interpreting data even from large studies. First, only 46 percent of the notified patients were tested. A study like this must be voluntary, and many patients may choose not to be tested or not to report their results. Second, since the date of the surgeon's HIV infection

is unknown, it is unknown what proportion of the patients tested were operated on by the surgeon while he was infected. Finally, even though none of the patients tested was positive, setting aside the I.V. drug user, the upper limit of the 95 percent confidence interval for the estimated rate of transmission is 0.5 percent or one in 200. That is, even if all patients in a large study are negative, we cannot statistically exclude the possibility that transmission could occur at a low rate.

In order to obtain precise estimates of patient risk from look-back studies, very large numbers of patients must be studied, probably in multiple investigations aggregated together with

[Add. 5]

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invasive procedures stratified according to degree of invasiveness. These investigations are frequently complicated by difficult confidentiality and liability issues. There are usually problems in documenting how long the surgeon or dentist may have been HIV positive, what infection-control precautions were actually used, and whether percutaneous injuries or breaks in technique may have occurred. If positive patients are identified, epidemiologic investigation of those patients for other risks and laboratory studies to sequence portions of the viral genome are not always technically possible.

Although additional patient look-back studies are needed, it seems unlikely that more precise estimates of patient risk will be available from look-back studies in the near future; therefore, we have attempted to estimate the risk to patients indirectly by constructing a model based on the limited data available.

When appropriate barrier precautions, that is, gloves, gowns and so on, are used, and recommended sterilization and disinfection procedures are followed, most cases of HIV transmission in health care settings are likely to result from percutaneous exposures to infected blood, such as may be caused,

[Add. 6]

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for example, by needlesticks or cuts with sharp objects.

The risk of HIV transmission from an infected surgeon or dentist to an uninfected patient due to a single percutaneous exposure of the patient to the health care worker's blood during an invasive procedure may be estimated as the product of the three probabilities listed on this slide, namely: (a), that the health care worker will sustain a sharp injury during the procedure; (b), that the sharp object causing the injury and now contaminated with the health care worker's blood will recontact the patient's open wound. This might happen, for example, if a surgeon or dentist were injured while working in a confined or poorly visualized anatomic location and the instrument subsequently touched the tissues of the patient. And finally, (c), the probability that infection would actually be transmitted to the patient after such an exposure.

In six prospective studies the percentage of surgical or obstetrical procedures during which at least one member of the surgical team experienced a sharp injury ranged from 1.3 to 15.4 percent. The variations among these rates may be due to differences in study methods, procedures observed and surgical

[Add. 7]

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techniques and precautions used. In these studies sharp injuries occurred on a regular basis during surgery despite a policy of universal precautions, at least in the United States hospitals. The proportion of these surgical procedures in which a patient may have been exposed to a health care worker's blood is unknown.

To obtain more information on the blood exposures during surgery, in 1990 CDC conducted a prospective observation study of operations at four hospitals, a large innercity hospital and a smaller suburban hospital in each of two high AIDS incidence areas in the United States. Trained observers who had no other duties in the operating room monitored a representative sample of 1,382 procedures during a nine-month period in five surgical specialties: general, orthopedic, gynecology, trauma and cardiac surgery. Data recorded by the observers included the nature, frequency and circumstances of health care worker contact with patient blood and also instances when a sharp object recontacted the patient's open wound after injuring the health care worker.

Preliminary results indicate that one or more sharp injuries occurred among surgical personnel during 6.9 percent of the operations observed,

[Add. 8]

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comparable to the previously reported studies. Ninety-nine injuries were observed, including 88 among surgeons. The rate of injury for an individual surgeon was 2.5 per 100 procedures performed. The sharp object causing the injury recontacted the patient's open wound in 32 percent of the 88 observed injuries in surgeons. Of the 28 injuries to surgeons

in which recontact was observed, 21 or 75 percent were caused by suture needles, three were caused by surgical wires, and one each was caused by a bone fragment, scissors, a staple gun and a retractor.

Using the data from this study in the model, we have here for column A the probability of a sharp injury to one surgeon during one procedure with the range there among the specialties, probability of recontact, and for the third parameter in the model the risk of infection transmission to a patient after a recontact exposure. This risk is unknown; therefore, we started by using data on the risk of infection transmission from a patient to a health care worker after a percutaneous exposure to infected blood.

In separate studies conducted by CDC and others, the probability of transmission after percutaneous exposure has been estimated to be

[Add. 9]

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approximately 0.3 percent for HIV-infected blood and approximately 30 percent for hepatitis B e-antigen-positive blood. E antigen is a marker of high infectivity in the blood of persons with hepatitis B infection. However, these numbers were derived primarily from studies of injuries with hollow-bore needles. Solid-bore suture needles might pose less risk than hollow-bore needles because less blood might be introduced into the wound. Also, passage of a sharp object through glove material may result in less blood exposure. Therefore, tenfold reductions in these values were also included in column C.

The probability of transmission from an HIV-positive or hepatitis B e-antigen positive surgeon to a single patient may be estimated by the product A, B, C from the previous slide. For HIV the probability ranges from .0024 percent, about one in 42,000, to .00024 percent, about one in 417,000 procedures. For e-antigen-positive surgeons the probability ranges from .24 percent, about one in 400, to .024 percent, about one in 4,000.

The cumulative probability that an infected surgeon will transmit infection to at least one patient has been estimated for one year, assuming that

[Add. 10]

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a surgeon may perform 500 procedures per year, and for a surgeon's remaining career after acquisition of HIV infection, assumed to be seven years. For example, if the risk of HIV transmission after a percutaneous exposure of a patient to an infected surgeon's blood is .3 percent, the probability that an HIV-infected surgeon will transmit HIV to at least one patient during one year of practice is 1.2 percent. The probability that the surgeon will transmit to at least one patient during the rest of his or her career is 8.1 percent. If the risk of transmission is .03 percent per single recontact, the probabilities are .12 percent during one year and .8 percent during seven years.

Data available to test the validity of these estimates are limited to a small number of hepatitis B investigations, all of which have a number of methodologic shortcomings. Also, these particular studies were done during an era when dentists rarely wore gloves. Among 1,640 procedures performed by surgeons, oral surgeons and dentists with either acute hepatitis B infection or who were e antigen carriers, hepatitis B

seroconversion was identified in one patient, for a rate of .06 percent. This is within the general range predicted by the model.

[Add. 11]

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Also according to this model, among the five surgical specialties studied, the highest risk to patients was for gynecology procedures. This is also supported by the hepatitis B experience. Aside from oral surgery, OB/GYN is the surgical specialty in which the most outbreaks of hepatitis B in patients of an infected surgeon have been reported. Of the 20 published hepatitis B outbreaks in patients of an infected worker, five outbreaks were linked to obstetricians and gynecologists.

We then estimated the number of patients who may theoretically have acquired HIV infection from infected surgeons in the United States based upon estimates of the number of surgeons with HIV infection, the number of invasive procedures performed by each surgeon while infected and the probability of transmission to a single patient. So you can see here we started with 42 AIDS cases reported, multiply eight to adjust for HIV infection, and multiplying by these additional factors yields the final result there.

Similar estimates for dentists are hampered by the lack of prospective studies of injuries during dental procedures. Available data permit only an indirect estimate. If a dentist performs approximately 3,000 procedures per year and has 12

[Add. 12]

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percutaneous injuries per year, as based on a questionnaire survey, the rate of sharp injury for a dentist is 12 divided by 3,000 or 0.4 per 100 procedures. Assuming that the percent of injuries in which the sharp object recontacted the patient is the same as for surgeons, 32 percent, the risk per procedure and the number of patients infected can be estimated in a similar manner. To date only a small fracture of this theoretical patient pool would have developed AIDS and been detected through AIDS surveillance.

As outlined in the draft document, this model and these estimates have several important limitations. Some of the limitations may result in overestimates of patient risk, others may result in underestimates, and still others may have effects in either direction. Perhaps the greatest overall limitation is the lack of data.

In summary, I would like to make three general points. Slide off, please. First, we recognize that there are many limitations to the model but we believe it is important to make a rough order-of-magnitude estimate of the risk of sporadic transmission to patients based on the data that are available. Second, whatever the risk is during a

[Add. 13]

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single procedure, the cumulative risk that at least one patient will be infected in an invasive procedure performed during the remaining career of an infected surgeon or dentist will be higher. Finally, although the three patients in Florida are the only ones now reported in which health-care-worker-to-patient transmission appears to have occurred, it seems likely that at

least some additional cases have occurred but have not been detected.

I will stop now for questions. Thank you.

DR. CURRAN: Thank you, Dr. Bell. Dr. Bell, thank you also for not exceeding your time allotted. Dr. Bell never exceeds his time allotted.

Joining Dr. Bell in the resource panel is Dr. Hal Margolis who is the Chief of the Hepatitis Branch of the Division of Viral and Rickettsial Diseases, Dr. Bill Martone and David Culber. Dr. Martone is the Director of the Hospital Infections Program at the Centers for Disease Control, and Dr. David Culver is the Chief of the Statistical Activity of that program. Dr. Don Marianos, a dental surgeon with the Dental Disease Activity, and Dr. Ruth Berkelman, the Chief of the Surveillance Branch responsible for AIDS surveillance and HIV infection reporting at CDC.

[Add. 14]

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DR. CURRAN: I think our interest at this meeting was to provide all even unpublished data, and this data is very new. It is a high priority for us to get more information. We have already alluded to the absence of information in the dental setting, and that's comparable.

Dr. Check.

DR. CHECK: Bill Check, Atlanta. Dr. Bell, you mentioned two pieces of cross-verification from real world data. You have another one that was just talked about

and I'm curious as to whether you have analyzed it. That is, given the assumptions and the predictions that you have made, can you calculate the probability for the Tennessee surgeon that, given 600 plus patients, what is the likelihood from your predictions that you would not see an infection? In other words, we are really asking about the power of measuring 600 patients and finding zero infections. Is that a meaningful result that contradicts anything else or is that something that is highly likely to see?

DR. CULVER: We haven't calculated that. I really can't answer that at this point.

DR. BELL: Only a couple of things to mention. One is that the statistical confidence

[Add. 15]

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interval on the experience in Tennessee does not rule out a transmission rate less than .5 percent.

DR. CURRAN: I might mention that Dr. Mishu is in the second row over there. You want to stand up. In case anyone wants to direct queries to her during the break or something.

On the left.

DR. VALACHOVIC: Richard Valachovic, Boston, Massachusetts. On the dentist side of the model in table two in which you come up with estimated number of patients infected, there are two pieces or two assumptions that you have made that I really have some problems with, because it really affects the power of the bottom line.

First of all, in the number of procedures that you use as an assumption per year of 3,000, you would base that on testimony from the ADA that there were potential exposures to blood. I just think that there is a big difference between 3,000 potential exposures to blood on the dentist side versus 500 surgical procedures on the surgeon side, and I don't think you are making an appropriate comparison. And so it comes down to the definition of what are you using as an invasive procedure.

DR. BELL: Let me clarify that. The

[Add. 16]

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risk per procedure was obtained for surgeons and dentists in very different ways. The risk per procedure for surgeons was based on observation studies. We had people standing there counting the injuries and the recontacts. And then we took the number of procedures that we knew they did per year and went from that route.

Now, for the dentists, there are no prospective studies. We didn't have anybody standing there counting. All we had was a figure that 12 injuries occurred per year. So in order to figure out the risk per procedure we had to look around and find a number for how many procedures were done in that year. And the ADA testimony to OSHA enabled us to come up with a number of about 3,000.

Now, the risk per procedure is then calculated as 12 divided by the 3,000. And we recognize that those procedures, most of them, are not nearly as invasive as a surgical procedure but it is a calculated value. And if somebody has a

number of procedures that is different -- for example, if you say the right number of procedures isn't 3,000, it is only 1,000, well, okay, then the risk per procedure is three times as high because it still has to come out

[Add. 17]

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with 12 injuries a year because that is the hard number we started with.

DR. VALACHOVIC: And then the 156 AIDS cases reported in dentists, is that in dentists or oral surgeons who are conducting invasive procedures or is it dental health workers as a common pool?

DR. BELL: That is another very good question. There are dental health care workers, and we don't know how many of them are dentists. As discussed in the document, the great majority of them are male and the great majority of dental ancillary personnel are female. So we just figured that we would use that number for dentists. It's an order of magnitude at best anyway.

DR. CURRAN: Dr. Marianos or Dr. Berkelman, do you want to comment?

MR. HOWARD: I am Richard Howard from the University of Florida. This assessment model is certainly a good first attempt, but because it could be used for phenomenal changes in policy decision, it is important that it be as accurate as possible. I have two areas of concern.

One is the implicit assumption that is made that the same volume of blood is transmitted by a suture needle which is not hollow as is transmitted by

[Add. 18]

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a hollow needle that has been used to calculate HIV transmission rates to health care workers.

The other is the recontact rate of 32 percent. Once a surgeon or member of the surgical team injures himself, the patient then is recontacted with that needle 32 percent of the time. That seems to be very high. And I wonder if part of the reason is that the data was collected in -- at least two of the hospitals were innercity hospitals where frequently residents are operating without appropriate staff supervision. And was the data the same between the innercity hospitals and the suburban hospitals?

DR. BELL: The first question about the hollow bore versus solid bore, I thoroughly agree with you. We don't know what the risk is, and I think we discussed that some.

The issue of the difference in injury rates and recontacting rates according to whether the operator was an attending physician or a resident physician has not been completely analyzed yet. But our first impression is that the injury rates were similar for the attendings and the house staff, and, at least looking at this quickly yesterday, it seemed that the recontacting rates were actually significantly higher for the attendings than for the

[Add. 19]

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house staff. That shouldn't be interpreted as any more than a simple statistical statement. We don't know. They may have been doing obviously much different procedures and so on. But I guess the answer to your question is it was not clearly apparent, for example, in fact, it seemed not to be true that all this was due to inexperienced residents.

FROM THE FLOOR: The reason for doing a statistical study like this is based on having seen a transmission occur. Yet out of a million transmissions in the United States, 100,000 deaths from AIDS, the only two places where we have seen this transmission is in one office, and the statistical evaluation of that office does not at all match up with your model to have three transmissions in one office. Yet, even based on that the one case doesn't match your model at all, there is a generalization of this to surgeons where we have still never seen a single transmission. And yet, even though there have been no transmissions, you come up with an estimation of there may have been a hundred such transmissions based on a projection where the numerator really is zero.

Because of this, I wonder when the CDC plans on doing another statistical theoretical

[Add. 20]

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evaluation of the risk of transmission of HIV from such things as mosquitoes, toilet seats or waiters.

DR. CURRAN: I think that is a very important comment. A risk assessment model is inherently risky, and it is true that the Florida investigation, as Dr. Schram had

previously commented, falls outside the bounds of the model. It would imply and the public would expect that the risk is far higher than Dr. Bell's model suggested by the observation of the single office with three patients.

Now, it also we think is an aberrancy. We think that, as with hepatitis B, this particular office and situation is likely to be an aberrancy. And the risk assessment model, although risky and subject to the criticism we hope to receive today and tomorrow, and I'm sure you won't disappoint us, is an attempt also to try to put this in some perspective; in a situation when we know the risk is not zero, to say to the public this risk we think falls within these kind of logical bounds, and we want to change these bounds with any data we can get or any theory we can get.

Middle, please.

MS. BARRETT: I'm Trish Barrett from the San Francisco Bay Area. I have been a full-time

[Add. 21]

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dropping it by a factor of ten. But I wonder in your observational study, when you looked at the instances of needlestick and percutaneous exposure, whether in fact many of those were trivial exposures. In my own experience I would certainly support the idea that needlestick injuries do occur, but most of them are indeed trivial with no blood at the site of puncture, perhaps only a puncture of the glove, et cetera. So I wonder whether that estimate also is even higher at the lower range of your estimate.

DR. BELL: Regarding your first question, you are correct in that the published studies to date report injury rates using the procedure as a denominator rather than the worker. The study that I presented did not do that. An injury occurred to at least one member of the surgical team in 6.9 percent of the cases; however, the risk for an individual surgeon procedure was 2.5 per 100 procedures. In other words, we did record the number of surgeons present and calculated the risk per surgeon.

Regarding the nature of the needlesticks that were observed, it's hard to define what was trivial. Certainly superficial needlesticks have resulted in HIV transmission, but we are still in the

[Add. 22]

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process of reviewing the data to try and see if we can characterize the injuries more fully.

DR. SECKINGER: Dan Seckinger, Chairman of the Florida Medical Association AIDS Activities. And I think in our discussion of both the case this morning and in discussion of the mathematical model, mention has been made that this is the only case in which transmission has occurred.

Now, over the months that we have been tracking this event with Dr. Witte and the Florida HRS people, I have been impressed with the role of established public health measures such as communicable disease reporting and contact tracing by name. These linkages might not be possible if it were not for that process. In states with greater prevalence, such as New

York and California, would demonstration of a case such as this have been possible?

DR. CURRAN: Dr. Berkelman.

DR. BERKELMAN: I think currently we have got AIDS case reporting in most states and not HIV infection reporting. So we are talking about investigation of AIDS cases that come to light with no identifiable risk. All of them are investigated. And most are reclassified.

[Add. 23]

Deposition of JOHN MOLINARI, Ph.D., August 17, 1995
[Page 32]

[by Mr. McCarthy]

Q. And you don't know of any evidence that supports the assertion that this was an intentional transmission by Dr. Acer?

A. There was a friend, who went public, of Dr. Acer a couple years ago who claimed that Dr. Acer had some ill feelings against people and wanted to publicize the AIDS epidemic. This has never been corroborated. In fact, my understanding is that when this person was questioned by the CDC, there were a number of holes in that story.

Q. It's correct, is it not, that HIV can be transmitted more readily from a patient to a dental care worker than it can from a dental care worker to a patient?

MR. KLEIN: Object to the form.

A. I would answer that the health care worker is generally the one with increased risk versus the patient when it comes to bloodborne disease.

Q. (BY MR. MCCARTHY) And why is that?

A. The health care worker is the one manipulating needles, sharp instruments, handling contaminated items in between patient care. The health care worker may be exposed to some sort of

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sharps or percutaneous injury if the patient moves or flinches unexpectedly during some surgical or other procedure, and generally they are the ones who are on the receiving end of these types of accidents.

Q. And that's why you say in your book that you wrote with Dr. Cottons and Dr. Terezhalmyn, "Practical Infection Control In Dentistry," that the danger is much greater for dentists and dental care workers than it is for patients. Am I correct?.

A. Correct.

Q. Just for the reasons you've just recited?

A. Correct.

[Add. 25]

CDC
Centers for Disease Control and Prevention

May 15, 1998/Vol. 47/No. RR-7

MMWR
Morbidity and Mortality Weekly Report

Recommendations and Reports

Public Health Service Guidelines for the Management of Health-Care Worker Exposures to HIV and Recommendations for Postexposure Prophylaxis

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention (CDC)
Atlanta, Georgia 30333

[Add. 26]

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PEP initiated at 72 hours had no effect (54). In macaques administered ZDV or BEA-005 1 to 72 hours after SIV intravenous challenge, earlier initiation of PEP was correlated with delayed onset and peak of antigenemia, decreased duration of antigenemia, and reduction in SIV serum titer; the most potent effect was evident when PEP was initiated within 8 hours of exposure (*43,56*). Studies in primates and murine and feline animal models have demonstrated that larger inocula decease prophylactic efficacy (*47,48,53,60*). In addition, delaying initiation, shortening the duration, or decreasing the antiretroviral dose of PEP, individually or in

combination, decreased prophylactic efficacy (*42,43,45,47,50,55*).

There is little information with which to assess the efficacy of PEP in humans. Seroconversion is infrequent after an occupational exposure to HIV-infected blood; therefore a prospective trial would need to enroll many thousands of exposured HCWs to achieve the statistical power necessary to directly demonstrate PEP efficacy. During 1987-1989, the Burroughs-Wellcome Company sponsored a prospective placebo-controlled clinical trial among HCWs to evaluate 6 weeks of ZDV prophylaxis; however this trial was terminated prematurely because of low enrollment (*61*). Because of current indirect evidence of PEP efficacy, it is unlikely that a placebo-controlled trial in HCWs would ever be feasible.

In the retrospective case-control study of HCWs, after controlling for other risk factors for HIV transmission, the risk of HIV infection among HCWs who used ZDV as PEP was reduced by approximately 81% (95% C1-43%-94%) (*23*). In addition, in a randomized, controlled, prospective trial (AIDS Clinical Trial Group [ACTG] protocol 076) in which ZDV was administered to HIV-infected pregnant women and their infants, the administration of ZDV during pregnancy, labor, and delivery and to the infant reduced transmission of 67% (*3*). Only 9%-17% (depending on the assay used) of the protective effect of ZDV was explained by reduction of the HIV titer in the maternal blood, suggesting that ZDV prophylaxis in part involves a mechanism other than the reduction of maternal viral burden (*26*).

The limitations of all of these studies must be considered when reviewing evidence of PEP efficacy. The extent to which data from animal studies can be extrapolated to humans is largely unknown, and the exposure route for mother-to-infant HIV transmission is not similar to occupational exposures; therefore these findings may not reflect a similar mechanism of ZDV prophylaxis in HCWs. Although the results of the retrospective case-control study of

HCWs suggest PEP efficacy, the limitations of that study include the small number of cases studied and the use of cases and controls from different cohorts.

Failure of ZDV PEP to prevent HIV infection in HCWs has been reported in at least 14 instances (*62-64*; G. Ippolito, AIDS Reference Center, Rome, Italy, and J. Heptonstall, Communicable Disease Surveillance Center, London, United Kingdom, personal communication, 1997). Although eight of the 13 source patients had taken ZDV, laboratory assessment for ZDV resistance of the virus from the source patient was performed in only three instances, two of which demonstrated reduced susceptibility to ZDV. In addition to possible exposure to a ZDV-resistant strain of HIV, other factors that may have contributed to the apparent failures in these instances may include a high titer and/or large inoculum exposure, delayed initiation and/or short duration of PEP, and possible factors related to the host (e.g., cellular immune system responsive

[Add. 27]

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The addition of a PI as a third drug for PEP following high-risk exposures is based on the site of activity in the replication cycle (i.e., after viral integration has occurred) and demonstrated effectiveness in reducing viral burden. Previously, indinavir (IDV) was recommended as the PI for PEP because of its increased bioavailability when compared with saquinavir and its more favorable immediate toxicity profile compared with ritonavir (*72*). In addition, requirements for dose escalation when initiating ritonavir make it less practical for use in PEP. Since the 1996 PEP recommendations were published, nelfinavir (NEL) was

approved for use by FDA and is now included in regimens recommended for the treatment of primary HIV infection (*69*). Also, FDA recently approved a soft-gel formulation of saquinavir (Fortovase™, manufactured by Hoffman-LaRoche, Inc., Nutley, New Jersey) that has improved bioavailability relative to its hard-gel formulation (Invirase™, manufactured by Hoffman-LaRoche, Inc.). However, the recommended dose of soft-gel saquinavir (1200 mg three times a day) is twice that of the hard-gel formulation (600 mg three times a day) and necessitates taking 18 pills a day, a factor that may influence HCW compliance if used for PEP. Based on these considerations, either IDV or NEL is recommended as first choice for inclusion in an expanded PEP regimen. If saquinavir is preferred by the prescribing physician, the soft-gel formulation (Fortovase™) should be used. Also, differences in the side effects associated with IDV and NEL, discussed below, may influence which of these agents is selected in a specific situation.

The NNRTIs (i.e., nevirapine and delavirdine) have not been included in these recommended regimens for PEP. As a class of antiretroviral agents, the NNRTIs are fast-acting and very potent, making them appealing in concept for PEP. In addition, there is some evidence of prophylactic efficacy (*73*). However, concerns about side effects and the availability of alternative agents argue against routinely using this class of drugs for initial PEP, although with expert consultation, an NNRTI might be considered.

Side Effects and Toxicity of Antiretroviral Agents

An important goal of PEP is to encourage and facilitate compliance with a 4-week PEP regimen. Therefore, the toxicity profile of antiretroviral agents, including the frequency, severity, duration, and reversibility of side effects, is a relevant consideration. All of the antiretroviral agents have been associated with side effects (See Appendix). However, studies of adverse events have been reported

primarily for persons with advanced disease (and longer treatment courses) and therefore may not reflect the experience of persons with less advanced disease or those who are uninfected (*74*). Side effects associated with many of the NRTIs (e.g., ZDV or ddI) are chiefly gastrointestinal (e.g. nausea or diarrhea), and in general the incidence of adverse effects has not been greater when these agents are used in combination (*72*).

All of the approved PIs may have potentially serious drug interactions when used with certain other drugs, requiring careful evaluation of concomitant medications being used by an HCW before prescribing a PI and close monitoring for toxicity when an HCW is receiving one of these drugs (See Appendix). PIs may inhibit the metabolism of nonsedating antihistamines and other hepatically metabolized drugs; NEL and ritonavir may accelerate the clearance of certain drugs, including oral contraceptives (requiring alternative or additional contraceptive measures for women taking these drugs.) The use of PIs also has been associated with new onset of diabetes mellitus,

[Add. 28]

hyperglycemia, diabetic ketoacidosis, and exacerbation of pre-existing diabetes mellitus (*75-77*), Nephrolithiasis has been associated with IDV use (including in HCWs using the drug for PEP) (*8*); however, the incidence of this potential complication may be limited by drinking at least 48 oz (1.5 L) of fluid per 24-hour period (e.g., six 8 oz glasses of water throughout the day) (*72*). Rare cases of hemolytic anemia also have been associated with the use of IDV. NEL, saquinavir, and ritonavir have been associated with the development of diarrhea; however, this side effect usually responds to treatment with antimotility agents that can be prescribed for us, if necessary, at the time any one of these drugs is

prescribed for PEP. The manufacturer's package insert should always be consulted for questions about potential drug interactions.

Among HCWs receiving ZDV PEP, usually at doses of 1,000-1,200 mg per day (i.e., higher than the currently recommended dose), 50%-75% reported one or more subjective (*7,78,79*). Common symptoms included nausea, vomiting, malaise or fatigue, headache, or insomnia. Mild decreases in hemoglobin and absolute neutrophil count also were observed. All side effects were reversed when PEP was discontinued.

Preliminary information about HCWs receiving combination drugs for PEP (usually ZDV plus 3TC with or without a PI) suggests that approximately 50%-90% of HCWs report subjective side effects that caused 24%-36% to discontinue PEP (*8-10*). One study documented that combination regimens that included ZDV at a lower dose (600 mg per day) were better tolerated than high-dose ZDV used alone (1,000-1,200 mg per day) (*10*). However, serious side effects, including nephrolithiasis, hepatitis, and pancytopenia, have been reported with the use of combination drugs for PEP (*9,80*; J.L. Gerberding, San Francisco General Hospital, personal communication, May 1997).

Resistance to Antiretroviral Agents

Known or suspected resistance of the source virus to antiretroviral agents, particularly to one or more agents that might be included in a PEP regimen, is a concern for those making decisions about PEP. Resistance of HIV has been reported with all available antiretroviral agents (*65*). However, the relevance of exposure to a resistant virus is not understood. Although transmission of resistant strains has been reported (*81-85*), in the perinatal clinical trial that studied vertical HIV transmission (ACTG protocol 076), ZDV prevented perinatal transmission despite genotypic resistance of HIV to ZDV in the mother (*66*). In addition, patients

generally take more than one antiretroviral drug and, unless testing is performed, often it is difficult to know to which drug(s) resistance exists. The complexity of this issue is further compounded by the frequency of cross-resistance within drug classes.

Resistance should be suspected in source patients when there is clinical progression of disease or a persistently increasing viral load and/or a decline in CDC T-cell count despite therapy, or a lack of virologic response to a change in therapy. Nevertheless, in this situation it is unknown whether a modification in the PEP regimen is necessary or will influence the outcome of an occupational exposure.

Antiretroviral Drugs in Pregnancy

Considerations for the use of antiretroviral drugs in pregnancy include their potential effect on the pregnant woman and on her fetus or neonate. The pharmacokinetics

[Add. 29]

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Assessment of Infection Risk

After an occupational exposure, the source-person and the exposed HCW should be evaluated to determine the need for HIV PEP. Follow-up for hepatitis B virus and hepatitis C virus infections also should be conducted in accordance with previously published CDC recommendations (*98,99*).

Evaluation of exposure. The exposure should be evaluated for potential to transmit HIV based on the type of body substance involved and the route and severity of the exposure. Exposures to blood, fluid containing visible blood, or other potentially infectious fluid (including semen; vaginal secretions; and cerebrospinal, synovial, pleural, peritoneal,

pericardial, and amniotic fluids) or tissue through a percutaneous injury (i.e., needlestick or other penetrating sharps-related event) or through contact with a mucous membrane are situations that pose a risk for bloodborne transmission and require further evaluation (Figure 1). In addition, any direct contact (i.e., personal protective equipment either was not used or was ineffective in protecting skin or mucous membranes) with concentrated HIV in a research laboratory or production facility is considered an exposure that requires clinical evaluation to assess the need for PEP.

For skin exposures, follow-up is indicated if it involves direct contact with a body fluid listed above and there is evidence of compromised skin integrity (e.g., dermatitis, abrasion, or open wound). However, if the contact is prolonged or involves a large area of intact skin, postexposure follow-up may be considered on a case-by-case basis or if requested by the HCW.

For human bites, the clinical evaluation must consider possible exposure of both the bite recipient and the person who inflicted the bite, HIV transmission only rarely has been reported by this route (*100,101*; CDC, unpublished data, 1998). If a bite results in blood exposure to either person involved, postexposure follow-up, including consideration of PEP, should be provided.

Evaluation and testing of an exposure source. The person whose blood or body fluids are the source of an occupational exposure should be evaluated for HIV infection. Information available in the medical record at the time of exposure (e.g., laboratory test results, admitting diagnosis, or past medical history) or from the source person may suggest or rule out possible HIV infection. Examples of information to consider when evaluating an exposure source for possible HIV infection include laboratory information (e.g., prior HIV testing results or results of immunologic testing [e.g.,

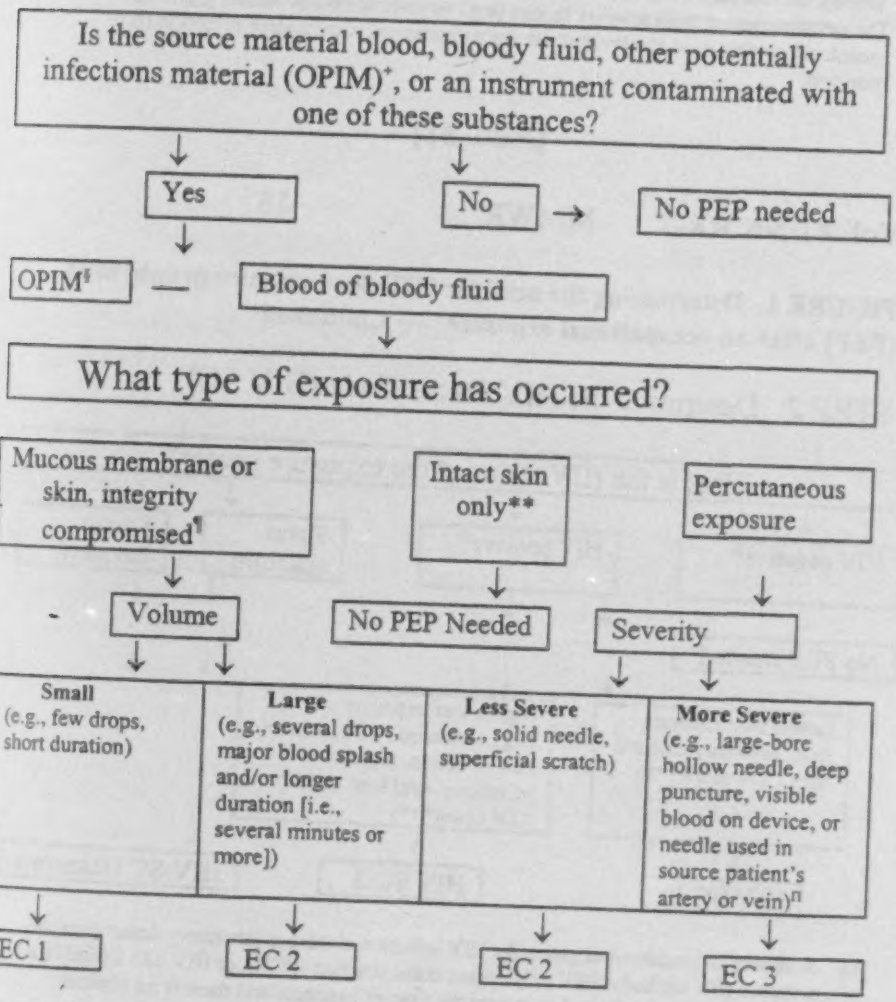
CD4+count]), clinical symptoms (e.g., acute syndrome suggestive of primary HIV infection or undiagnosed immunodeficiency disease), and history of possible HIV exposures (e.g., injecting-drug use, sexual contact with a known HIV-positive partner, unprotected sexual contact with multiple partners [heterosexual and/or homosexual], or receipt of blood or blood products before 1985).

If the source is known to have HIV infection, available information about this person's stage of infection (i.e., asymptomatic or AIDS), CD4+ T-cell count, results of viral load testing, and current and previous antiretroviral therapy, should be gathered for consideration in choosing an appropriate PEP regimen. If this information is not immediately available, initiation of PEP, if indicated, should not be delayed; changes in the PEP regimen can be made after PEP has been started, as appropriate.

[Add 30]

FIGURE 1. Determining the need for HIV postexposure prophylaxis (PEP) after an occupational exposure*

STEP 1: Determine the Exposure Code (EC)



* This algorithm is intended to guide initial decisions about PEP and should be used in conjunction with other guidance provided in this report.

† Semen or vaginal secretions; cerebrospinal, synovial, pleural, peritoneal, pericardial, or amniotic fluids; or tissue.

§ Exposures to OPIM must be evaluated on a case-by-case basis. In general, these body substances are considered a low risk for transmission in health-care settings. Any

unprotected contact to concentrated HIV in a research laboratory or production facility is considered an occupational exposure that requires clinical evaluation to determine the need for PEP.

¶ Skin integrity is considered compromised if there is evidence of chapped skin, dermatitis, abrasion, or open wound.

** Contact with intact skin is not normally considered a risk for HIV transmission. However, if the exposure was to blood, and the circumstance suggests a higher volume exposure (e.g., an extensive area of skin was exposed or there was prolonged contact with blood), the risk for HIV transmission should be considered.

Π The combination of these severity factors (e.g., large-bore hollow needle and deep puncture) contribute to an elevated risk for transmission if the source person is HIV-positive.

[Add. 31]

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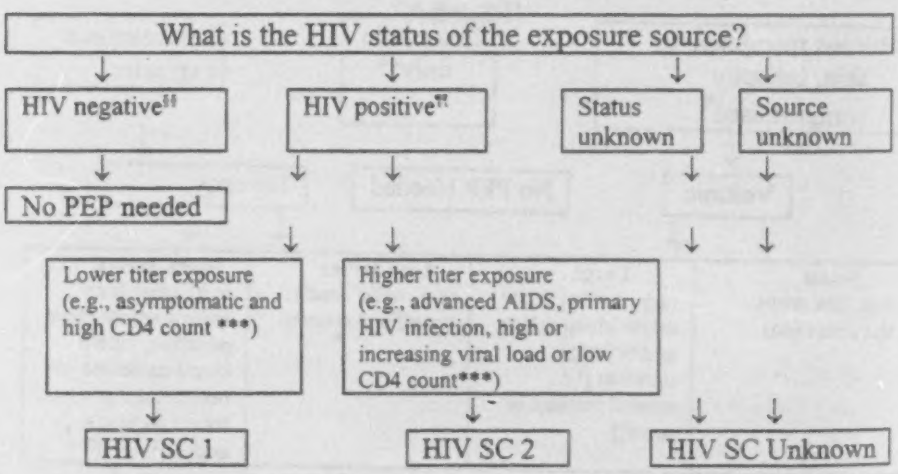
FIGURE 1. Determining the need for HIV postexposure prophylaxis (PEP) after an occupational exposure* — Continued

STEP 2: Determine the HIV Status Code (HIV SC)



§§ A source is considered negative for HIV infection if there is laboratory documentation of a negative HIV antibody, HIV polymerase chain reaction (PCR), or HIV p24 antigen test result from a specimen collected at or near the time of exposure and there is no clinical evidence of recent retroviral-like illness.

¶¶ A source is considered infected with HIV (HIV positive) if there has been a positive laboratory result for HIV antibody, HIV PCR, or HIV p24 antigen or physician-diagnosed AIDS.

*** Examples are used as surrogates to estimate the HIV titer in an exposure source for purposes of considering PEP regimens and do not reflect all clinical situations that may be observed. Although a high HIV titer (HIV SC 2) in an exposure source has been associated

with an increased risk for transmission, the possibility of transmission from a source with a low HIV titer also must be considered.

STEP 3: Determine the PEP Recommendation

EC	HIV SC	PEP recommendation
1	1	**PEP may not be warranted.** Exposure type does not pose a known risk for HIV transmission. Whether the risk for drug toxicity outweighs the benefit of PEP should be decided by the exposed HCW and treating clinician.
1	2	**Consider basic regimen.**[∏] Exposure type poses a negligible risk for HIV transmission. A high HIV titer in the source may justify consideration of PEP. Whether the risk for drug toxicity outweighs the benefit of PEP should be decided by the exposed HCW and treating clinician.
2	1	**Recommended basic regimen.** Most HIV exposures are in this category; no increased risk for HIV transmission has been observed but use of PEP is appropriate.
2	2	**Recommended expanded regimen.**[§§§] Exposure type represents an increased HIV transmission risk.
3	1 or 2	**Recommended expanded regimen.** Exposure type represents an increased HIV transmission risk.
Unknown		If the source or, in the case of an unknown source, the setting where the exposure occurred suggests a possible risk for HIV exposure and the EC is 2 or 3, consider PEP basic regimen.

∏ Basic regimen is four weeks of zidovudine, 600 mg per day in two or three divided doses, and lamivudine, 150 mg twice daily.

§§§ Expanded regimen is the basic regimen plus either indinavir, 800 mg every 8 hours, or nelfinavir, 750 mg three times a day.

[Add. 32]

If the HIV serostatus of the source person is unknown, the source person should be informed of the incident and, if consent is obtained, tested for serologic evidence of HIV infection. If consent cannot be obtained (e.g., patient is

unconscious), procedures should be followed for testing source persons according to applicable state and local laws. Confidentiality of the source person should be maintained at all times.

HIV-antibody testing of an exposure source should be performed as soon as possible. Hospitals, clinics, and other sites that manage exposed HCWs should consult their laboratories regarding the most appropriate test to use to expedite these results. An FDA-approved rapid HIV-antibody test kit should be considered for use in this situation, particularly if testing by enzyme immunoassay (EIA) cannot be completed within 24-48 hours. Repeatedly reactive results by EIA or rapid HIV-antibody tests are considered highly suggestive of infection, whereas a negative result is an excellent indicator of the absence of HIV antibody. Confirmation of a reactive result by Western blot or immunofluorescent antibody is not necessary for making initial decisions about postexposure management but should be done to complete the testing process.

If the source is HIV seronegative and has no clinical evidence of acquired immunodeficiency syndrome (AIDS) or symptoms of HIV infection, no further testing of the source is indicated. It is unclear whether follow-up testing of a source who is HIV negative at the time of exposure, but recently (i.e., within the last 3-6 months) engaged in behaviors that pose a risk for HIV transmission, is useful in postexposure management of HCWs; HCWs who become infected generally seroconvert before repeat testing of a source would normally be performed.

If the exposure source is unknown, information about where and under what circumstances the exposure occurred should be assessed epidemiologically for risk for transmission of HIV. Certain situations, as well as the type of exposure, may suggest an increased or decreased risk; an important consideration is the prevalence of HIV in the population group (i.e., institution or community) from which the contaminated

source material is derived. For example, an exposure that occurs in a geographic area where injecting-drug use is prevalent or on an AIDS unit in a health-care facility would be considered epidemiologically to have a higher risk for transmission than one that occurs in a nursing home for the elderly where no known HIV-infected residents are present. In addition, exposure to a blood-filled hollow needle or visibly bloody device suggests a higher-risk exposure than exposure to a needle that was most likely used for giving an injection. Decisions regarding appropriate management should be individualized based on the risk assessment.

HIV testing of needles or other sharp instruments associated with an exposure, regardless of whether the source is known, is not recommended. The reliability and interpretation of findings in such circumstances are unknown.

Clinical Evaluation and Baseline Testing of Exposed HCWs

Exposed HCWs should be evaluated for susceptibility to bloodborne pathogen infections. Baseline testing (i.e., testing to establish serostatus at the time of exposure) for HIV antibody should be performed. If the source person is seronegative for HIV, baseline testing or further follow-up of the HCW normally is not necessary. If the source person has recently engaged in behaviors that are associated with a risk for HIV transmission, baseline and follow-up HIV-antibody testing (e.g., 3 and/or 6 months

[Add. 33]

postexposure) of the HCW should be considered. Serologic testing should be made available to all HCWs who are concerned that they may have been exposed to HIV.

For purposes of considering HIV PEP, the evaluation also should include information about medications the HCW

may be taking and any current or underlying medical conditions or circumstances (i.e., pregnancy, breast feeding, or renal or hepatic disease) that may influence drug selection. Pregnancy testing should be offered to all nonpregnant women of childbearing age whose pregnancy status is unknown.

HIV PEP

The following recommendations apply to situations where an HCW has had an exposure to a source person with HIV or where information suggests that there is a likelihood that the source person is HIV-infected. These recommendations are based on the risk for HIV infection after different types of exposure and limited data regarding efficacy and toxicity of PEP. Because most occupational HIV exposures do not result in the transmission of HIV, potential toxicity must be carefully considered when prescribing PEP. When possible, these recommendations should be implemented in consultation with persons having expertise in antiretroviral therapy and HIV transmission.

Explaining PEP to HCWs

Recommendations for chemoprophylaxis should be explained to HCWs who have sustained occupational HIV exposures (Figure 1). For exposures for which PEP is considered appropriate, HCWs should be informed that a) knowledge about the efficacy and toxicity of drugs used for PEP are limited; b) only ZDV has been shown to prevent HIV transmission in humans; c) there are no data to address whether adding other antiretroviral drugs provides any additional benefit for PEP, but experts recommend combination drug regimens because of increased potency and concerns about drug-resistant virus; d) data regarding toxicity of antiretroviral drugs in persons without HIV infection or in pregnant women are limited for ZDV and not known regarding other antiretroviral drugs; and e) any or all drugs for PEP may be declined by the HCW. HCWs who have HIV occupational

exposures for which PEP is not recommended should be informed that the potential side effects and toxicity of taking PEP outweigh the negligible risk of transmission posed by the type of exposure.

Factors in Selection of a PEP Regimen

Selection of the PEP regimen should consider the comparative risk represented by the exposure and information about the exposure source, including history of and response to antiretroviral therapy based on clinical response, CD4+ T-lymphocyte counts, viral load measurements, and current disease stage. Most HIV exposures will warrant only a two-drug regimen, using two NRTIs, usually ZDV and 3TC. The addition of a third drug, usually a PI (i.e., IDV or NEL), should be considered for exposures that pose an increased risk for transmission or where resistance to the other drugs used for PEP is known or suspected.

[Add. 34]

Timing of PEP Initiation

PEP should be initiated as soon as possible. The interval within which PEP should be started for optimal efficacy is not known. Animal studies have demonstrated the importance of starting PEP within hours after an exposure (*43,54,56*). To assure timely access to PEP, an occupational exposure should be regarded as an urgent medical concern and PEP started as soon as possible after the exposure (i.e., within a few hours rather than days). If there is a question about which antiretroviral drugs to use, or whether to use two or three drugs, it is probably better to start ZDV and 3TC immediately than to delay PEP administration. Although animal studies suggest that PEP probably is not effective when started later than 24-36 hours postexposure (*42,55,56*), the

interval after which there is no benefit from PEP for humans is undefined. Therefore, if appropriate for the exposure, PEP should be started even when the interval since exposure exceeds 36 hours. Initiating therapy after a longer interval (e.g., 1-2 weeks) may be considered for exposures that represent an increased risk for transmission; even if infection is not prevented, early treatment of acute HIV infection may be beneficial (*69*). The optimal duration of PEP is unknown. Because 4 weeks of ZDV appeared protective in HCWs (*2*), PEP probably should be administered for 4 weeks, if tolerated.

PEP if Serostatus of Source Person is Unknown

If the source person's HIV serostatus is unknown at the time of exposure (including when the source is HIV negative but may have had a recent HIV exposure), use of PEP should be decided on a case-by-case basis, after considering the type of exposure and the clinical and/or epidemiologic likelihood of HIV infection in the source (Figure 1). If these considerations suggest a possibility for HIV transmission and HIV testing of the source is pending, it is reasonable to initiate a two-drug PEP regimen until laboratory results have been obtained and later modify or discontinue the regimen accordingly.

PEP is Exposure Source is Unknown

If the exposure source is unknown, use of PEP should be decided on a case-by-case basis. Consideration should include the severity of the exposure and the epidemiologic likelihood that the HCW was exposed to HIV.

PEP for Pregnant HCWs

If the HCW is pregnant, the evaluation of risk and need for PEP should be approached as with any other HCW who has had an HIV exposure. However, the decision to use any antiretroviral drug during pregnancy should involve discussion between the woman and her health-care provider regarding the potential benefits and potential risks to her and her fetus.

Follow-up of HCWs Exposed to HIV

Postexposure Testing

HCWs with occupational exposure to HIV should receive follow-up counseling, postexposure testing, and medical evaluation regardless of whether they receive PEP. HIV-antibody testing should be performed for at least 6 months postexposure (e.g., at

[Add. 35]

6 weeks, 12 weeks, and 6 months). It is unclear whether an extended follow-up period (e.g., 12 months) is indicated in certain circumstances. Although rare instances of delayed HIV seroconversion have been reported (*36,37*, J.L. Gerberding, San Francisco General Hospital, unpublished data, May 1997), the infrequency of this occurrence does not warrant adding to HCWs' anxiety by routinely extending the duration of postexposure follow-up. Circumstances for which extending the duration of follow-up have been suggested include the use of highly potent antiretroviral regimens (i.e., more than two drugs) because of theoretical concerns that HIV seroconversion could be delayed, or simultaneous exposure to HCV. Data are insufficient for making a general recommendation in these situations. However, this should not preclude a decision to extend follow-up in an individual situation based on the clinical judgment of the HCW's health-care provider. HIV testing should be performed on any HCW who has an illness that is compatible with an acute retroviral syndrome, regardless of the interval since exposure. HIV-antibody testing using EIA should be used to monitor for seroconversion. The routine use of direct virus assays (e.g., HIV p24 antigen EIA or polymerase chain reaction for HIV

RNA) to detect infection in exposed HCWs generally is not recommended (*34*). Although direct virus assays may detect HIV infection a few days earlier than EIA, the infrequency of HCW seroconversion and increased costs of these tests do not warrant their routine use in this setting. Also, HIV RNA is approved for use in established HIV infection; its reliability in detecting very early infection has not been determined.

Monitoring and Management of PEP Toxicity

If PEP is used, drug-toxicity monitoring should be performed at baseline and again 2 weeks after starting PEP. Clinical judgment, based on medical conditions that may exist in the HCW and any toxicity associated with drugs included in the PEP regimen, should determine the scope of testing. Minimally these should include a complete blood count and renal and hepatic chemical function tests. Monitoring for evidence of hyperglycemia should be included for HCWs whose regimen includes any PI; if the HCW is receiving IDV, monitoring for crystalluria, hematuria, hermolytic anemia, and hepatitis also should be included. If toxicity is noted, modification of the regimen should be considered after expert consultation; further diagnostic studies may be indicated.

HCWs who fail to complete the recommended regimen often do so because of the side effects they experience (e.g., nausea and diarrhea). These symptoms often can be managed without changing the regimen by prescribing antimotiliy and antiemetic agents or other medications that target the specific symptoms. In other situations, modifying the dose interval (i.e., administering a lower dose of drug more frequently throughout the day, as recommended by the manufacturer), may help promote adherence to the regimen.

Counseling and Education

Although HIV infection following an occupational exposure occurs infrequently, the emotional impact of the exposure often is substantial (*102,103*). In addition, HCWs

are given seemingly conflicting information. Although HCWs are told that there is a low risk for HIV transmission, a 4-week regimen of PEP is recommended and they are asked to commit to behavioral measures (i.e., sexual abstinence or condom use) to

[Add. 36]

prevent secondary transmission, all of which influence their lives for several weeks to months (102). Therefore, access to persons who are knowledgeable about occupational HIV transmission and who can deal with the many concerns an HIV exposure may raise for the HCW is an important element of postexposure management.

HIV-exposed HCWs should be advised to use the following measures to prevent secondary transmission during the follow-up period, especially during the first 6-12 weeks after the exposure when most HIV-infected persons are expected to seroconvert; use sexual abstinence or condoms to prevent sexual transmission and to avoid pregnancy; and refrain from donating blood, plasma, organs, tissue, or semen. If the exposed HCW is breastfeeding, she should be counseled about the risk for HIV transmission through breast milk, and discontinuation of breastfeeding should be considered, especially following high-risk exposures. If the HCW chooses to receive PEP, temporary discontinuation of breastfeeding while she is taking PEP should be considered to avoid exposing the infant to these agents. NRTIs are known to pass into breast milk; it is not know whether this also is true for PIs.

There is no need to modify an HCW's patient-care responsibilities to prevent transmission to patients based solely on an HIV exposure. If HIV seroconversion is detected, the

HCW should be evaluated according to published recommendations for HIV-infected HCWs (*104*).

Exposed HCWs should be advised to seek medical evaluation for any acute illness that occurs during the follow-up period. Such an illness, particularly if characterized by fever, rash, myalgia, fatigue, malaise, or lymphadenopathy, may be indicative of acute HIV infection but also may be due to a drug reaction or another medical condition.

Exposed HCWs who choose to take PEP should be advised of the importance of completing the prescribed regimen. Information should be provided about potential drug interactions and the drugs that should not be taken with PEP, the side effects of the drugs that have been prescribed (See Appendix), measures to minimize these effects, and the methods of clinical monitoring for toxicity during the follow-up period. They should be advised that the evaluation of certain symptoms should not be delayed (e.g., back or abdominal pain, pain or urination or blood in the urine, or symptoms of hyperglycemia [i.e., increased thirst and/or frequent urination]).

RECOMMENDATIONS FOR THE SELECTION OF DRUGS FOR PEP

The selection of a drug regimen for HIV PEP must strive to balance the risk for infection against the potential toxicity of the agent(s) used. Because PEP is potentially toxic, its use is not justified for exposures that pose a negligible risk for transmission (Figure 1). Also, there is insufficient evidence to recommend a highly active regimen for all HIV exposures. Therefore, two regimens for PEP are provided (Table 1): a "basic" two-drug regimen that should be appropriate for most HIV exposures and an "expanded" three-drug regimen that should be used for exposures that pose an increased risk for transmission (Figure 1) or where resistance to one or more antiretroviral agents is known or suspected. When possible, the regimens should be implemented in

consultation with persons having expertise in antiretroviral treatment and HIV transmission.

[Add. 37]

TABLE 1. Basic and expanded postexposure prophylaxis regimens

Regimen Category	Application	Drug regimen
Basic	Occupational HIV exposures for which there is a recognized transmission risk (Figure 1)	4 weeks (28 days) of both zidovudine 600 mg every day in divided doses (i.e., 300 mg twice a day, 200 mg three times a day or 100 mg every 4 hours) **and** lamivudine 150 mg twice a day.
Expanded	Occupational HIV exposures that pose an increased risk for transmission (e.g., larger volume of blood and/or higher virus titer in blood) (Figure 1).	Basic regimen plus **either** indinavir 800 mg every 8 hours or nelfinavir 750 mg three times a day.*

*Indinavir should be taken on an empty stomach (i.e., without food or with a light meal) and with increased fluid consumption (i.e., drinking six 8 oz glasses of water throughout the day); nelfinavir should be taken with meals.

Situations That Require Special Consideration

Resistance of the Source Virus to Antiretroviral Drugs

It is unknown whether drug resistance influences transmission risk; however, transmission of drug-resistant HIV has been reported (*81,82*) and is therefore a theoretical concern when choosing PEP regimens. If the source-person's virus is known or suspected to be resistant to one or more of the drugs included in the PEP regimen, the selection of drugs to which the source person's virus is unlikely to be resistant is recommended (*69*). If the resistance is to one class of antiretroviral drugs, the addition to the basic PEP regimen of a drug from another class might be considered (e.g., addition of a PI when a source patient has not been treated with a PI but has virus resistant to one or more NRTIs). It is strongly

recommended that PEP be started regardless of the resistance status in the source virus; if resistance is known or suspected, a third of fourth drug may be added to the regimen until consultation with a clinical expert in the treatment of HIV infection or disease can be obtained.

[Add. 38]

OVERSIGHT HEARINGS ON OSHA'S PROPOSED STANDARD TO PROTECT HEALTH CARE WORKERS AGAINST BLOOD-BORNE PATHOGENS INCLUDING THE AIDS AND HEPATITIS B VIRUSES

Volume 1

HEARINGS
BEFORE THE
SUBCOMMITTEE ON HEALTH AND SAFETY
OF THE
COMMITTEE ON EDUCATION AND LABOR
HOUSE OF REPRESENTATIVES
ONE HUNDRED FIRST CONGRESS
FIRST SESSION

HEARINGS HELD IN WASHINGTON DC, NOVEMBER 2, 9, AND 15, 1989

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John F. Storm
Vice President, Government Affairs

Dear Ms. Brooke, September 27, 1989

Enclosed is a copy of the transcript of the 60 MINUTES broadcast that you requested.

If we can be of further assistance on this or any other matter, please let us know.

Sincerely,

s/John F. Storm

Ms. Suzanne Brooke
Subcommittee on Health and Safety
B-345A Rayburn House Office Building
U.S. House of Representatives
Washington, D.C. 20515-6103

Encls

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From 60 Minutes
9/24/89

gible jail time, and the Mob's revenge, when, after two years of illness, he died in a Cleveland hospital of brain cancer.

WILLIAM MCCARTHY: I call him a crooked stool pigeon. He lies.

REASONER *[voice over]:* William McCarthy, Jackie Presser's successor as president of the Teamsters, was cross-examined last spring by a Senate subcommittee about Presser's allegations in his F.B.I. informant file that McCarthy was controlled by La Cosa Nostra - by the Mob.

Mr. MCCARTHY: I have never been approached by Cosa Nostra. You have Williams and Presser -- one is a schemer and one was a liar, and they both were mixed up, as I say, with the Cosa Nostra by their own admission.

Mr. RAVEL: As long as organized crime, particularly the La Cosa Nostra, has its tentacles in organized labor, the base information that Jackie Presser gave will be used as a predicine for further investigations and to show the

pattern of activities that has lead the Mob to control several elements of labor.

REASONER: Dead but not forgotten.

Mr. RAVEL: That's right.

REASONER: Today, in Washington, D.C., an administrator appointed by a federal judge supervises the headquarters of the International Brotherhood of Teamsters. His job is to root out officials still controlled by the Mob.

[Commercial break]

Dr. Day

CROFT: The effects of the AIDS epidemic on the health care system are just beginning to be felt. That's there's a shortage of hospital beds for people with AIDS is well-known. What's not well-known is that there may soon be a shortage of doctors and nurses to treat people with AIDS. The plain fact is, one out of five of the teaching hospitals in this country have reported staff members quitting for fear of contracting the virus.

According to Dr. Lorraine Day, a respected surgeon and university professor, those fears are justified. For more than a year she has waged a one-woman crusade warning health care workers about the risks of getting infected, and attacking the medical establishment for underplaying the threat. This month, Dr. Day became the highest-ranking defector, resigning her job as Chief of Orthopedic Surgery at San Francisco General Hospital.

Dr. LORRAINE DAY: I'm quitting because the risk is too high to continue to operate on so many patients who are either AIDS infected or HIV infected.

CROFT: You're afraid of getting AIDS.

Dr. DAY: I think that's a legitimate fear.

CROFT *[voice over]*: Dr. Lorraine Day is so afraid that she dons a spacesuit every time she operates. It was originally developed to protect the patient from the doctor. Dr. Day wears it to protect herself from the patient, or more specifically, from the patient's blood, which in many hospitals is now considered a toxic substance.

[Interviewing] Have you calculated your chances?

Dr. DAY: Someone else described my risk as being 12 percent per year, and 49 percent at the end of five years, of turning positive from occupational exposure.

CROFT: A 50 percent chance of getting AIDS within five years?

Dr. DAY: That's what they estimate.

CROFT: You think that performing a hip replacement operation is high-risk behavior.

Dr. DAY: Certainly in an HIV positive patient, or a high-risk patient. It is high-risk behavior. It seems to be as high-risk behavior as people having anal sex.

CROFT *[voice over]*: She's been called a homophobic bigot by San Francisco's gay community, an hysterical scaremonger by some of her colleagues. But Lorraine Day has found an audience. She is a sought after speaker with medical

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groups around the country, which want to hear her message -- that the medical establishment has endangered lives by underplaying the risks of on-the-job AIDS infection.

Dr. JOHN LUCE [?]: I think that there's an excitement about Dr. Day, and perhaps there's a little bit of scariness to all of this that is appealing to people the way that people watch horror movies.

CROFT *[voice over]*: Dr. John Luce is a former Chief of Staff at San Francisco General, and one of Dr. Day's adversaries in a debate that has turned doctor against doctor in a hospital which has been in the forefront of fighting the AIDS epidemic, which has already had one nurse infected on the job.

Dr. LUCE: I think that having fears about HIV infection and AIDS is very appropriate, and I think any physician who doesn't have fears about getting infected is being crazy. At the same time, I think to carry the fear to the point that Dr. Day does, and to try to instill, and I believe that she has instilled, fear in other people, is wrong. I think that Dr. Day is an alarmist.

CROFT *[voice over]*: Dr. Day is accustomed by now to being called an alarmist. She says it usually comes from people who don't understand the operating room.

Dr. DAY: Before we knew about AIDS, I would have blood all over my face and on my mask. We got blood in our eyes frequently. We get blood on our hands all the time because our gloves get torn. We change them frequently, but every surgeon has blood in their gloves, in contact with their hands, many times before they are aware of it.

CROFT *[on camera]*: This is the first time in 40 years, since the polio epidemic, that doctors have had to face personal danger on a daily basis, and it's beginning to have a profound impact on the profession. Dr. Day is not alone in her fear.

EMERGENCY ROOM DOCTOR: This is a male, about late 30s. He was gunshot by a 38 Special --

CROFT *[voice over]*: In an operating room at San Francisco General, where a team of doctors was working on an emergency gunshot victim, we

found everyone wearing some kind of eye protection, and the surgeon in charge, Dr. Bill Schecter [?], was wearing wading boots.

[Interviewing] Do you know if that blood was infected with the AIDS virus?

Dr. BILL SCHECTER: I assume every patient that I treat is infected. If I have a scratch on my foot, and I'm standing in blood, we know that viral transmission can occur.

Dr. LUCE: If any one of you gets stuck with a needle in the middle of the night, there's a hotline to call, someone at the end of the hotline is going to give you AZT if you choose it, they're going to draw your blood --

CROFT *[voice over]*: Punctures from needles, both suture and hypodermic, are the most common kind of accident with contaminated blood. When doctors or nurses get stuck, their blood is rushed to the lab for testing. The Centers for Disease Control places the chances of someone getting the virus from a contaminated needle at one in 200.

Dr. DAY: A couple of my residents have been punctured with known HIV positive blood in the last couple of months.

CROFT: How common are needle sticks in this hospital?

DR. LUCE: They're very common. I would imagine that the average house officer, by which I mean an intern or a resident in training, probably has a needle-stick injury at least every couple of weeks, if not weekly.

CROFT: How common are needle pricks with HIV positive blood.

DR. LUCE: Well, if you are an intern working on the medical service, who's taking care of patients on the AIDS ward, all of whom, by definition, have AIDS, and you're getting stuck every two weeks, and those patients make up a third, a quarter of your practice, your chances of getting stuck are very good.

Dr. DAY: Our risk is one in 100 for single needle-stick with AIDS blood, and it

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can be the first one. It doesn't take 200. And I ask you, if you came to work every day and flipped the lightswitch on in your office, and only one out of 200 times you were electrocuted, would you consider that low risk?

CROFT: The people at this hospital, for the most part, the administrators, say that you are suffering, essentially, from what they call AIDS anxiety.

Dr. DAY: Oh, well, yes I am suffering from AIDS anxiety. I think that if you were faced with a fatal disease every single day when you went to

work, I think that you would have a certain amount of anxiety. It is appropriate. It's called survival.
CROFT *[on camera]*: There are six and a half million health care workers in the United States, and according to the Centers for Disease Control here in Atlanta, there are only 18 documented cases of on-the-job infection. Those numbers have been used to try and persuade health care workers that their chances of getting the virus on the job are low.
Dr. DAY: How did they come up with those figures? Did they test all 6.5 million health care workers? They tested a very small percentage, probably less than five percent, and they haven't tested them themselves.
CROFT: You think the CDC is underplaying the threat.
Dr. DAY: Absolutely, Absolutely.
CROFT *[voice over]*: In fact, no one really believes the figure of 18 health care workers infected, including Dr. Jim Curran [?] of the CDC.
Dr. JIM CURRAN: It's certainly much more than 18, and probably much less than several hundred.
CROFT *[voice over]*: Dr. Curran is in charge of the AIDS program at the Centers for Disease Control. He acknowledges that relatively few health care workers have been tested, but insists the documented infection rate is small. He does concede some workers face more risks that others.
Dr. CURRAN: I think that surgeons, and people who have a substantial amount of needle-sticks and cuts during surgery, have considerable potential of infection from blood-born infections, including HIV, as well as Hepatitis B.
CROFT: We asked the question to Lorraine Day, and she said, "Absolutely. It's high-risk behavior. My chances of contracting the AIDS virus from one needle-stick are greater than they are from having anal sex or gay sex."
Dr. CURRAN: They're probably about the same. A needle-stick from an AIDS patient versus one sexual contact with anal intercourse.
CROFT: So you're saying the risks are pretty high. Significant.
Dr. CURRAN: I think the risks are something that have to be dealt with, that are significant.
CROFT *[voice over]*: Most health care workers we spoke with were under the impression that the CDC does not consider the risk significant, and as recently as April of 1988, the CDC published a report which warned health care workers to be careful with blood, but also reported that the occupational risk of acquiring HIV in a health care setting is low.
DR. CURRAN: I don't think that characterizing the risk as low is accurate. I believe that the risk for someone who gets a needle-stick is a substantial one, and one that should be concerned.
CROFT: Are you guilty of underplaying the risks to medical workers?
DR. CURRAN: I don't like to dwell on the past so much as to deal with what has to happen in the future. I don't feel any guilt, but if someone

says that the risk has been minimized, whether it's been done by the CDC or by someone else, if the perception out there is misunderstanding on the part of health care workers of their own risk, misunderstanding of how they can minimize the risk, then it's the CDC's job, and the health community's job, to make them understand it.

CROFT *[voice over]*: Lorraine Day says the best way to clear up misunderstanding is to do more testing, and not just of doctors. She is perhaps the foremost proponent in the country for testing of hospital patients.

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HIV/AIDS
Surveillance Report

U.S. HIV and AIDS cases reported through June 1994
Mid-year Edition Vol. 6, No. 1

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[Add. 42]

Table 11. Health-care workers with documented and possible occupationally acquired AIDS/HIV infection, by occupation, reported through June 1994, United States[1]

Occupation	Documented occupational transmission[2] No.	Possible Occupational transmission[3] No.
Dental worker, including dentist	-	7
Embalmer/morgue technician	-	3
Emergency medical technician/paramedic	-	9
Health aide/attendant	1	9
Housekeeper/maintenance worker	1	6
Laboratory technician, clinical	15	14
Laboratory technician, nonclinical	2	1
Nurse	13	19
Physician, nonsurgical	6	9
Physician, surgical	-	1
Respiratory therapist	1	2
Technician, dialysis	1	1
Technician, surgical	2	1
Technician/therapist, other than those listed above	-	4
Other health-care occupations	-	2
Total	42	88

[1]Health-care workers are defined as those persons, including students and trainees, who have worked in a health-care, clinical, or HIV laboratory setting at any time since 1978. See MMWR 1992;41:823-5.
[2]Health-care workers who had documented HIV seroconversion after occupational exposure or had other laboratory evidenced of occupational infection: 36 had percutaneous exposure, 4 had mucocutaneous exposure, 1 had both percutaneous and mucocutaneous exposures, and 1 had an unknown route of exposure. Thirty-eight exposures were to blood from an HIV-infected person, 1 to visibly bloody fluid, 1 to an unspecified fluid, and 2 to concentrated virus in a laboratory. Fifteen of these health-care workers developed AIDS.
[3]These health-care workers have been investigated and are without identifiable behavioral or transfusion risks; each reported percutaneous or mucocutaneous occupational exposures to blood or body fluids, or laboratory solutions containing HIV, but HIV seroconversion specifically resulting from an occupational exposure was not documented.

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[JAMA, July 17, 1991-Vol 266, No. 3]

Letters----------

Do Universal Precautions Reduce Needlestick Injuries?

To the Editor.-- The article by Wong et al[1] documents reductions in occupational exposure to blood and body fluids among physicians after the implementation of universal precautions (UPs). However, the investigators have misinterpreted their data relating to needlestick injuries. Although they found a 62% reduction in needlestick injuries when UPs were in effect, their data show that this difference was not statistically significant (P= .123, Mantel-Haenszel χ^2). This is not surprising in view of the small number of needlestick injuries reported during the study. Despite a lack of statistical significance, the investigators inappropriately concluded that "the implementation of UPs

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should reduce the risk of occupational human immunodeficiency virus (HIV) infection by the same rate [as needlesticks] (62%). . . ." Based on the study data alone, the correct conclusion is that the implementation of UPs should *not* be expected to reduce either needlestick injuries or the risk of HIV infection associated with them.

Even if the reported difference had been statistically significant, the result would still be inconclusive because the investigators have used an inappropriate denominator (physician-patient-care months) for calculating needlestick rates. The risk of needlestick injuries is directly related to the number and types of needles used,[2] and only indirectly, if at all to the duration of patient care. The use of patient-care months for the calculation of needlestick rates among physicians is misleading. For example, in this study, physicians could

achieve an apparent reduction in needlestick rates simply by delegating a greater proportion of needle procedures to nursing staff.

There is a growing body of evidence indicating that UPs alone are not sufficient to reduce the risk of needlestick injuries.[3-5] We are concerned that prolonging the reliance on UPs to prevent needlestick injuries will only further delay the transition to safer needle designs, a delay that will be measured by the number of health care workers contracting fatal, preventable disease.

Janine Jagger, MPH, Ph.D
Richard D. Pearson, MD
University of Virginia
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Charlottesville

1. Wong ES Stotka JL, Chinchilli VM, Williams DS, Stuart GC, Markowitz SM. Are universal precautions effective in reducing the number of occupational exposures among health care workers? JAMA. 1991;265:1123-1128.
2. Jagger J, Hunt EH, Brand-Elnaggar J, Pearson RD. Rates of needle-stick injury caused by various devices in a university hospital. N Engl J Med. 1988;319:284-288
3. Ribner BS. Landry MN, Gholson GL, Linden LA. Impact of a rigid, puncture resistant container system upon needlestick injuries. *Infect Control.* 1987;8:63-66.
4.Krasinski K, LaCouture R, Holzman R.S. Effect of changing needle disposal systems on needle puncture injuries. *Infect Control.* 1987;8:59-62.
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In Reply. -- We believe our choice of patient-care months as the denominator for occupational exposures is neither inappropriate nor misleading. In our study, we were interested in the difference in the frequency of occupational exposures before and after implementation of UPs and, specifically, with regard to needlestick injuries, the percent reduction as a result of UPs (ie, the rate of injuries after implementation divided by

the rate of injuries before implementation). For this calculation, it is clear that the choice of denominators is irrelevant since they cancel out. We contend that the risk of needlestick injuries is procedure-dependent and the number of patients cared for on the study wards is the most important determinant of the number of procedures performed. Since we observed no variations in the month-to-month patient census or any changes in ward practices during the study period, we attributed the reduction in the frequency of exposures to the effect of UPs. Thus, while it is possible that physicians could have actually reduced their risk of needlestick injuries by delegating procedures to nurses after implementation of UPs, we have no reason to believe that this occurred and, in fact, this was not observed.[1]

Regarding our interpretation of needlestick data, we documented a 62% reduction in the frequency of needlestick injuries after the implementation of UPs. Although this was not statistically significant, we believe the observed reduction in injuries among our physicians reflects a real trend that would be statistically significant had our study period been extended. Studies on the epidemiology of needlestick injuries suggest that placement of puncture-resistant needle disposal units at the site of use can prevent certain types of needlestick injuries, namely those due to recapping, transport of uncapped needles, and improper disposal.[2,3] These types of preventable injuries represent approximately 20% of all needlestick injuries reported by hospital employees. However, studies also suggest that among house-staff physicians, the percentage of preventable needlestick injuries may be much higher than among nonphysician health care workers, eg, nurses.[4,5] In our recent study,[4] the proportion of injury among house-staff physicians due to recapping was 54% and due to improper disposal, 3%. Thus, the placement of needle disposal units in treatment areas may have a greater effect on physicians than on other health care workers, which may explain why our study, which observed physicians only, demonstrated a

benefit, while other studies, which have included all groups of health care workers, did not.

As studies by Jagger and others have shown, the problem of needlestick injuries is complex. The solution, by necessity, will also be complex and involve multiple approaches, including education, better needle disposal systems, and improved design of needles and related equipment. Jagger and Pearson misinterpret our study in that we are not suggesting that UPs, with improved access to needle disposal, will by themselves solve the needlestick injury problem. Our results do suggest that UPs are helpful and, as such, should be included in any prevention program.

Edward S. Wong, MD
Sheldon M. Markowitz, MD
Hunter Holmes McGuire
Veterans Affairs
Medical Center
Richmond, VA

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5. Mangione CM Gerberding JL, Cummings SR. Occupational exposure to HIV: frequency and rates of underreporting of percutaneous and mucocutaneous exposures by medical housestaff. *Am J Med*. 1991;90:85-90.

[partial letter captioned "Medical Malpractice Suits and Autopsies" omitted]

[Add. 49]

Safer Needle Devices: Protecting Health Care Workers
http://www.osha-slc.gov/SLTC/needle...ces/saferneedledevices.ntm1#orohiv

Department of Labor
United States of America

Safer Needle Devices: Protecting Health Care Workers

Prepared by the Occupational Safety and health Administration
Directorate of Technical Support
Office of Occupational Health Nursing
October 1997

Acknowledgment:

This document was developed by the Office of Occupational Health Nursing, with significant contributions from Susan Elliott, ARNP, MSN, MPH, OSHA Nurse Intern, and Digna Walker, RN, B.S.N., OSHA Nurse Intern.

Contents

Acronyms and Abbreviations

Purpose of this Packet

Epidemiology of Needlestick Injuries

Occupational Risk of Needlestick Injuries
- Occupational Risk of Hepatitis B
- Occupational Risk of Hepatitis C
- Occupational Risk of HIV

Definition of Safer Needle Device

[Add. 50]

Safer Needle Devices: Protecting Health Care Workers
http://www.osha-slc.gov/SLTC/needle...ces/saferneedledevices.ntm1#orohiv

(Source: Ippolito, 1997)

Q *How serious is the occupational risk of needlestick injuries to health care workers?*

A One of the most critical control components of health care worker protection against bloodborne pathogens must be the reduction of sharps-related incidents. The statistics cited below provide a picture of the seriousness of the problem.

- Hospital-based healthcare workers experience some **800,000 needlestick injuries** each year in the United States (Jagger, 1990). **About 2% or approximately 16,000** of these are likely to be comtaminated by HIV (American Hospital Association, 1991). **Needlestick injuries account for up to 80%** of all accidental exposures to blood (Jagger, 1988).
- Based on various studies, researchers have documented that **needlestick injuries are under reported** by health care workers and the number of exposures could potentially be much higher (Hamory, 1983). Chiarello (1992) cites several studies that found rates of under-reporting between 40.4% and 53% for nurses and 92% for laboratory personnel. Physicians under reported needlestick injuries by 70% to 95%.
- In well-documented studies, injuries from contaminated needles and other sharp devices used in the health care setting have been associated with transmission of bloodborne pathogens to health care workers. In fact, **more than 20 pathogens have been transmitted through sharps or needlestick injuries** (Chiarello, 1992). Of these, HIV, HBV and HCV pose the greatest risk to the health care worker (Ippolito et al., 1997). The risk of transmission of HBV and HCV through percutaneous injury is much higher than for HIV (CDC, 1991).
- Understanding the prevalence of these bloodborne diseases in the patient population gives a better picture of the risk health care workers face:
    - ◊ A study of the Johns Hopkins Hospital emergency room determined the prevalence of the HCV, HBV and HIV in blood samples from 2523 patients. Of these patients:

◊

- **18% were seropositive for HCV**
- **5% were seropositive for HBV**
- **6% were seropositive for HIV (Kalen et al, 1997).**

◊ **An estimated 1.25 million people** in the U.S. are chronically infected with **HBV** and 6,000 die each year as a result of HBV-related liver disease (Moyer & Hodgson, 1996).

◊ In 1989, the CDC studied the prevalence of HIV in three inner city hospitals and three suburban hospitals. In the three inner city hospitals, seroprevalence of HIV ranged from 4.1 to 8.0 per 100 patients. The suburban hospitals' seroprevalence rates ranged from 0.2 to 6.1 per 100 patients (Marcus et al., 1989)

◊ In 1997, the National Institutes of Health estimated that nearly 4 million people in the U.S. are infected with Hepatitis C (NIH, 1997). Of these, 85% develop chronic HCV infection and the potential for transmission of HCV to others (CDC, 1997).

Q ***What occupational risk does Hepatitis B pose to the health care worker?***

A For more than 50 years, HBV infection, a well-documented and recognized occupational hazard, has been and continues to be **one of the most common bloodborne pathogens among health care workers.** Studies conducted prior to implementation of recommendations to prevent bloodborne pathogen

Studies conducted prior to implementation of recommendations to prevent bloodborne pathogen transmission (1976-1985) show that health care workers had a **prevalence of HBV infection three to five times higher** than the general U.S. population (Moyer & Hodgson, 1996).

- **Hepatitis B is much more transmissible than HIV.** Health care workers at greatest risk work in areas where they are directly exposed to blood (in emergency rooms, clinical laboratories, operating rooms, hemodialysis units, etc.) (CDC, 1991).
- The risk of a health care worker contracting HBV from needlestick injuries ranges from **6% to 30%** (CDC, 1991).
- In 1994, **1000 health care workers developed HBV infection,** and each year **100 to 200 health care workers die** from this disease (CDC, 1997). The Centers for Disease Control and prevention (CDC) estimates that the annual number of new HBV infections in health care workers has steadily declined from 12,000 in 1985, due in part to the widespread adoption of universal precautions, vaccination against hepatitis B and the implementation of OSHA's Bloodborne Pathogens Standard (Moyer & Hodgson, 1996).

Q ***What occupational risk does Hepatitis C pose to the health care worker?***

A **Hepatitis C virus infection** is a major cause of chronic liver disease in the United States and worldwide. The virus, because of its similarity to HBV, ***presents an occupational risk*** to persons whose work activities involve handling human blood and body fluids (CDC, 1997).

- **Needlestick injuries** are the most common cause of **occupational HCV exposure** (Hibberd, 1995).
- In 1995, an estimated **560 to 1,120 cases of HCV infection occurred among health care workers** who were occupationally exposed to blood (Alter, 1993).
- **No vaccine is available for hepatitis C** and no effective post-exposure prophylaxis is known at this time (CDC, 1997).

- **Screening tests for hepatitis C** antibodies are commercially available, but interpretation of the results, especially in a post-exposure situation, is limited by several factors.
- A positive result does not distinguish between acute, chronic, or past infection, and a negative result does not indicate the absence of acute infection, only the absence of antibodies to HCV.
- False positives are common in populations with a low prevalence of HCV.
- The tests do not detect HCV antibodies in approximately 5% of people (CDC, 1997).
- As many as **85% of all HCV-infected persons develop chronic infection.** Persons with chronic hepatitis are at increased risk for cirrhosis and primary hepatocellular carcinoma. Hepatitis C is now the leading reason for liver transplantation in the United States (NIH, 1997).

Q ***What occupational risk does HIV pose for the health care worker?***

A HIV infection has been reported after occupational exposures to HIV-infected blood through **needlesticks or cuts; splashes in the eyes, nose, or mouth; and skin contact.**

- **Exposures from needlesticks or cuts cause most infections.** The average risk of HIV infection after a needlestick exposure to HIV-infected blood is **0.3% or 1 in 300.** Even though the risk of seroconversion after needlestick is relatively rare, injured health care workers may suffer disabling physical side effects from post-exposure anti-viral medication as well as severe emotional trauma as they await their test results (CDC, 1991).
- The risk after exposure of the eye, nose, or mouth to HIV-infected blood is estimated to be, on the average, **0.1% or 1 in 1,000** (CDC, 1987).
- The risk after exposure of the skin to HIV-infected blood is estimated to be less than 0.1%. The

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Safer Needle Devices: Protecting Health Care Workers
http://www.osha-slc.gov/SLTC/needle...ces/saferneedledevices.ntml#orohiv

The risk after exposure of the skin to HIV-infected blood is estimated to be less than 0.1%. The risk may be higher if the skin is broken or if the contact involves a large area of skin or is prolonged (CDC, 1987).

- As of July, 1996, CDC had received reports of **52** documented cases and ***111*** "possible" cases (i.e., documentation was unclear) of occupationally acquired HIV among health care workers in the United States. **Of the 52 documented cases of occupationally acquired HIV infection, 45 resulted from needle sticks or cuts (CDC HIV/AIDS Surveillance Report, 1996)** (Table 2). The number of occupationally acquired HIV infections in health care workers is increasing each year (Figure 3).

[Table 2 and Figure 3 omitted]

[Add. 53]

Safer Needle Devices: Protecting Health Care Workers
http://www.osha-slc.gov/SLTC/needle...ces/saferneedledevices.ntml#orohiv

[balance of Figure 3 omitted]

Note: The CDC did not publish statistics on occupationally acquired HIV in health care workers in the years 1989, 1990, and 1991. The CDC began publishing statistics on health care workers with possible occupationally acquired HIV in 1992.

Q ***Why are universal precautions and personal protective equipment not adequate to protect the health care worker against needlestick injuries?***

A Using universal precautions, along with personal protective equipment, engineering controls and other work practice controls, reduces employee exposure to bloodborne pathogens. Personal

protective equipment provides a barrier to protect skin and mucous membranes from contact with blood and other potentially infectious material (OPIM), but **most personal protective equipment is easily penetrated by needles.** Needlestick injuries are caused by unsafe needle devices rather than careless use by health care workers. (Jagger, 1988). Safer needle devices have been shown to significantly reduce the incidence of accidental needlesticks and exposure to potentially fatal bloodborne illnesses (CDC, 1997).

Q ***What is a safer needle device?***

A A **safer needle device incorporates engineering controls** to **prevent needlestick injuries** before, during, or after use through built-in safety features. The term, **"safer needle device,"** is broad and includes many different types of devices from those that have a protective shield over the needle to those that do not use needles at all. **The common feature of effective safer needle devices is that they reduce the risk of needlestick injuries for health care workers.**

Q ***Can safer needle devices prevent needlestick injuries?***

A All needlestick injuries are not preventable, but research has shown that almost 83% of injuries from

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A
hollowbore needles can be prevented (Ippolito et al, 1997). **Many of these needlesticks can be prevented by using devices that have needles with safety features or eliminate the use of needles altogether** (e.g., needleless IV connectors, self re-sheathing needles, and blunted surgical needles).

Most current research is hospital based and studies have indicated that a significant portion of needlestick injuries occur when manipulating IV lines or administering IV and IM injections (Jagger, 1988). In 1992 the FDA published a safety alert warning of the risk of needlestick injuries from the use of hypodermic needles as a connection between two pieces of IV equipment. This alert was based on research that demonstrated that secondary IV tubing with connector needles was associated with the highest risk of needlestick injury. The use of needleless IV systems or systems with recessed needles to connect adjoining equipment was strongly encouraged in this alert (See Appendix A).

Two new studies indicate that the use of safer needle designs can reduce the risk of needlestick injuries among health care workers. The National Center for Infectious Diseases (NCID) Hospital Infections Program conducted the studies in collaboration with eight hospitals in three U.S. cities. The results appear in the January 17, 1997 issue of MMWR. (See References)

The first study describes the use of blunt suture needles during gynecologic surgery, and indicates that blunt suture needles may reduce the likelihood of a needlestick during surgery by as much as 86%. The second study examined the use of safer needles for drawing blood and found that safer needles for drawing blood may reduce needlesticks to health care workers by 27% to 76%. The investigations also found that the use of safer needles did not lessen the quality of patient care. Further, the safer needles were generally accepted by health care workers.

Some devices have not been well-accepted in the clinical setting nor associated with a significant decease in injury rate. These results may be explained by lack of training or lack of support for change in the clinical setting (Chiarello, 1992).

Although all major medical device manufacturers market devices with safety features, **no standard criteria exist for evaluating the safety claims of these features.** Employers implementing needlestick prevention programs should evaluate the effectiveness of various devices in their specific settings.

Q *What are the design features of a safer needle device?*

A The Food and Drug Administration (FDA, 1992, 1995) has suggested that a safety feature designed to protect health care workers should:

- Provide a barrier between the hands and the needle after use;
- The safety feature should allow or require the worker's hands to remain behind the needle at all times;
- Be an integral part of the device and not an accessory;
- Be in effect before disassembly and remain in effect after disposal to protect downstream workers; and
- Be simple and self evident to operate and require little or no training to use effectively.

Features designed to protect the health care workers should not "have a negative impact on the delivery of patient care. As an example, there are a few reports of increased bloodstream infections with the use of needleless IV systems" (Chiarello, 1997).

Q *What are some of the types of safety features used in safer needle devices?*

A The types of safety features used in safer needle devices can be categorized according to certain

[Add. 55]

A The types of safety features used in safer needle devices can be categorized according to certain aspects of the safety feature, i.e., whether the feature is active or passive and whether the engineering control is part of the device (Chiarello, 1995).

- **Passive safety features** remain in effect before, during and after use; health care workers do not have to activate them. Passive features enhance the safety design and are more likely to have a greater impact on prevention.
- **Active devices** require the health care worker to activate the safety mechanism. Failure to do so leaves the worker unprotected. Proper use by health care workers is the primary factor in the effectiveness of these devices.
- An **integrated safety design** means that the safety feature is built in as an integral part of the device and cannot be removed. This design feature is preferred.
- An **accessory safety device** is a safety feature that is external to the device and must be carried to or temporarily or permanently fixed to the point of use. This design also is dependent on employee compliance and according to some researchers, is not desirable.

(See Appendix B for examples of ways these engineering features can be designed into needle devices.)

Q ***How can health care employers evaluate and select safer needle devices?***

A OSHA's Bloodborne Pathogens Standard requires that "Each employer having an employee(s) with occupational exposure ... shall establish a written Exposure Control Plan designed to eliminate or minimize employee exposure" [29 CFR 1910.1080)(c)(1)(ii)(B)]. A variety of safer needle devices is now widely available. Manufacturers have responded to the need for safer devices and as a result, a wave of safer medical products have flooded the marketplace. One thousand U.S. patents for safer medical devices have been issued since 1984 (Ippolito, 1997). Employers are faced with the tremendous task of selecting and evaluating products from the vast array of devices available.

Although OSHA does not require employers to institute the most sophisticated engineering controls, it does require the employer to evaluate the effectiveness of existing controls and to review the feasibility of instituting more advanced engineering controls (CPL

2-2.44C). OSHA staff should encourage an employer to implement a needlestick prevention program. Research to date has shown that **no single safer needle device will work equally well in every facility** so employers must develop their own programs to select the most appropriate devices. The goal is to **choose devices that are:**

- **Clinically effective,**
- **Acceptable to user, and**
- **Most effective in reducing needlestick injuries in that particular setting** (Chiarello, 1995).

To evaluate and select appropriate safer needle devices, health care employers should review available needlestick injury data including the personnel involved, the devices used, and the circumstances and frequency of needlestick events. This information can assist the employer in determining how employees can maximally benefit from a product change to a safer needle devices. Although not required by OSHA, **collection and evaluation of complete needlestick injury data by hospitals are key to identifying injury patterns and then implementing an effective abatement plan (Chiarello, 1995).**

Q ***What are the steps a health care employer should consider in developing a comprehensive needlestick prevention program and implementing safer needle devices?***

A Chiarello (1995) describes a framework to aid health care employers in establishing a comprehensive program to select and evaluate safer medical devices in a systematic manner. The

[Compend Contin Educ Dent, Vol. XIV, No. 8, p. 966 (Aug. 1993)]

Dental Infection Control Forum

By John A. Molinari, PhD

Infected Health-Care Professionals: Healers or Modern Day Lepers?
Part 2--Approaches and Recommendations

In the first part of this two-part series, basic concerns were discussed regarding the potentially volatile issue of health-care workers infected with the human immunodeficiency virus (HIV). These concerns included health-care workers' occupational risks for HIV, patient risks during treatment by HIV-infected health-care workers, and possible ramifications of proposed legislation designed to identify infected health-care workers and require informed consent before rendering patient care. This follow-up article will focus on approaches and recommendations that attempt to address the problem by using science-based knowledge as a foundation.

One theme that should always be considered when modification of public-health policy is contemplated includes two components: 1) health-care risks must first be demonstrated; and 2) proposed solutions must be deliberated in light of their own expected benefits and risks. Most people would agree that issues surrounding HIV infection and the acquired immunodeficiency syndrome (AIDS) are more emotionally charged than any other recently encountered infectious disease. Additional concern was exhibited by the public in 1990, when the first of a total of six HIV-infected patients in a Florida dental practice were determined to have been infected by a dentist who carried the same HIV strain. Perceived patient risks of HIV transmission continue to provide segments of the media with opportunities to make

undocumented claims. It is difficult to change perceptions, especially where safety and risk issues are concerned. One can only respond by offering data from ongoing look-back and epidemiological investigations. These indicate that more than 19,000 patients of HIV-infected health-care workers have been tested, and none were found to have been infected by health-care workers.[1] In light of the perception-vs-data debate, what can be done, then, to protect the high standards of patient care, give reasonable assurances to patients, and, at the same time, acknowledge the rights of health-care workers?

In 1991, two states, New York and Michigan, drafted and approved guidelines for HIV-infected health-care workers, which were later modified to include workers infected with the hepatitis B virus (HBV).[2,3] Both documents were subsequently reviewed by the Centers for Disease Control and Prevention (CDC) and deemed to be equivalent to the CDC "Recommendations for Preventing Transmission of the Human Immunodeficiency Virus and the Hepatitis B Virus During Exposure-Prone Invasive Procedures."[4] Table 1 lists the specific recommendations included in the Michigan guidelines. These guidelines were based on ongoing studies that indicate an extremely low risk for HIV transmission and a low risk of HBV cross-infection from infected health-care workers to patients. A fundamental principle for provision of patient care is strict adherence to the application of universal precautions to prevent transmission of blood-borne infections. When the data and historical success of appropriate infection-control procedures are considered, mandatory HIV testing of all health-care workers and limiting the practice of HIV- or HBV-infected health-care workers are simply not justified.

Can we guarantee zero risk for both the health-care workers and patients? No--however, in acting responsibly toward fulfilling our professional role as oral-care providers, it is incumbent on each health-care worker to invest time, effort, and active participation in those educational aspects of disease prevention that can provide the greatest measure of personal

protection to the health-care worker and to the patient population. Reduction of risk factors in both professional and personal arenas remains the cornerstone in conscientious and responsible actions designed to guarantee absolute minimal risk of infection. The time, effort, and even dollars invested toward the pursuit of minimal risk is the most responsible option available to the health-care worker to provide both personal and patient confidence in establishing minimal disease risk.

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[Add. 57]

IN THE UNITED STATES DISTRICT COURT
DISTRICT OF MAINE
----oOo----

SIDNEY ABBOTT, Plaintiff	)	
vs.	)	CIVIL ACTION No. 97-0273-B
RANDON BRAGDON, DMD Defendant	)	

Deposition of
DEBORAH GREENSPAN
Tuesday, July 25, 1995

Reported by:
MARLENE LEE, CSR No. 4623

[Pages 2 and 3 omitted]

[Page 4]

DEBORAH GREENSPAN,
having been duly sworn, testified as follows:

EXAMINATION BY MR. McCARTHY

MR. McCARTHY: Q. Would you state your full name for the record.

A. Deborah Greenspan.

Q. And your age?

A. Fifty-five.

Q. Present occupation?

A. Clinical professor of oral medicine in the Department of Stomatology, School of Dentistry, University of California.

Q. Is that your full-time employment?

A. Yes, it is.

Q. Do you treat patients in that capacity?

A. Yes, I do.

Q. To what extent?

A. I see patients in the Stomatology Clinical Center several days a week.

Q. Would you say you spend most of your time seeing patients?

A. I would say approximately 50 percent of my time is spent seeing patients.

Q. Are you those patients' primary dental

[Add. 58]

***

[page 17 omitted]

[Page 18]

A. No.

Q. Is there a greater risk of transmission of HIV from patient to health care provider than from health care provider to patient, in your opinion?

MS. PERLEY: What type of health care provider are you talking about?

THE WITNESS: Could you repeat the question so I'm clear?

MR. McCARTHY: Q. Sure. Is there a greater risk of the transmission of HIV from patient to health care provider than from health care provider to patient?

A. There is a difference.

Q. What is the difference?

A. There are no documented cases of patients acquiring HIV infection from the health care provider.

Q. But what is the answer to my question?

A. If you repeat the question. I think I'd answered it.

Q. Is there a greater risk of transmission of HIV from health care provider to patient than there is from patient to health care provider?

A. There is no risk -- there is no

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documented risk reported of a patient acquiring HIV infection from a health care provider. There are a number of cases that have been documented of HIV being acquired by a health care worker as a result of exposure.

Q. And so what is the answer to my question?

A. That there is a difference in risk between the two.

Q. And which risk do you see as greater?

A. The risk is greater of acquiring -- the risk is very small, but nevertheless it is greater to acquire HIV infection from a patient to a health care provider than the other way around. But it is very small.

Q. You made reference to the term "documented." Do you think it's possible to believe transmissions have taken place of HIV, whether they be from health care provider to patient or vice-versa, without the transmission being documented?

A. Not that I would use to make any judgments from. I would only use documented evidence.

Q. And when you say "documented," in terms

[page 20 omitted]

[Add. 59]

[pages 21, 22, 23 and 34 omitted]

[Add. 60]

****

[pages 153, 154, 155, and part of page 156 omitted]

[Page 156]

.....

Q. On Pages 100 and 182 of your book, "Aids and the Mouth," you state that the general dentist gives an average of 177 local injections a month, and that the rate of needle-stick injuries was 1.5 per 100 injections.

A. That was based, as far as I remember, on a survey that had been done.

Q. Was that a reliable survey?

A. Based upon the reliability of self-report, then I would say that the reporting was reliable. But it's always based on -- if it's based

[Add. 61]

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on self-report, it's based on the ability of people to recall.

But the survey, I trust -- but there was limitations to such surveys.

Q. Do you think that the figure of 1.5 needle-stick injuries per 100 injections is a reliable statistic for scientists to utilize in considering HIV issues?

A. I'm not sure how it pertains to HIV issues. But in answer directly to your question, I think that there is one survey and gives us some idea of how, in the context of that survey, that was what was reported And therefore I believe it was reliable.

Q. Do you think it's fair for one who is trying to estimate the rate of needle-stick injuries to rely on this calculation of 1.5 per 100 injections that you have referred to in your book?

A. I think that this was one survey that was done and gives one an idea of the number that were reported in this instance.

Q. But do you have any reason to discount the survey?

A. I have no reason to discount the survey. Merely that it must be taken in context of

[Page 158]

subsequent surveys that are done.

Q. Have you heard of subsequent surveys that would cause you to believe the estimate of sticks per injection should be different than 1.5 per 100?

A. There has been a subsequent survey after this book was published where the numbers are slightly different.

Q. How?

A I don't recall what they were, but I would comment that they are slightly different, as often happens in many surveys.

Q. When you say "slightly different," they might be 1.6? You don't mean they might be 5.5 per injection?

A. I believe they are within the same, if I may use the term, ball park.

Q. How do you define "injection" in the sense you utilized it in the sentence we just read?

A. Can you remind me of what we said? Sorry.

Q. I think you said, "General dentists give an average of 177 local anesthetic injections per month, and the rate of needle-stick injuries is 1.5 per 100 injections."

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A. "Injection" merely follows the term "local anesthetic." It's part of the same process. The local anesthetic is what you put in. Injection is the way you deliver it.

[rest of page 159 and page 160 omitted]

[Add. 62]

[Compend Contin Educ Dent, Vol XIV, No. 6, p. 706]

Dental Infection Control Forum

By John A. Molinari, PhD

Infected Health-Care Professionals: Healers or Modern Day Lepers? Part I--Issues and Considerations

The working environments of health-care providers changed dramatically on July 27, 1990. It was on this date that the Centers for Disease Control and Prevention (CDC) reported the first instance of possible transmission of the human immunodeficiency virus (HIV) to a patient from an HIV-infected health-care worker. The public and many others in the health professions were stunned, and soon the case of the "Florida dentist" was being discussed and debated in many forums. Subsequent information linking five additional patients to the dentist's HIV strain added to the turmoil and emerging public concern. Unfortunately, the exact mode of viral transmission has not been delineated to date, and probably never will be elucidated.

As a result of extensive media attention and the absence of important epidemiologic information, many health professionals, and the public, have drawn conclusions that are based more on frustration, fear, and anger than on scientific data. It is not my purpose to review the known details of this case in this article. No matter what happened, the results have already been tragic to those infected, their families, and to society in general. Instead, in the first part of this two-part series, I will offer my views on some important issues that have the potential to polarize treatment providers from their patients. These issues are: occupational HIV risks for health-care workers, mandatory HIV testing, and notification of HIV status to patients.

The following discussion will briefly outline known health-care worker occupational risks, policy dilemmas, and possible ramifications of proposed legislation aimed at practice limitations for infected health-care workers. Part 2 of this article will discuss the specific approaches and recommendations that have been generated by groups of health professionals, their organizations, and a number of state health departments.

There is one question that has been on the minds of health-care workers since the earliest reports of patients with acquired immunodeficiency syndrome (AIDS) were published in 1981. That question is: how much of an occupational risk do we have when treating patients with AIDS? Subsequent isolation and characterization of HIV provided opportunities to use serological assays to investigate suspected cross-infection cases. Numerous studies have been reported and continue to be performed to examine possible incidents of HIV transmission in a variety of clinical settings. Despite thousands of reported accidental exposures, 31 occupational HIV transmissions were documented by the CDC, as of July 1992, with 65 additional cases still under investigation (Table 1).[1]

A few important trends become apparent while reviewing these data: (1) while not zero, the risk to health-care workers appears to be very small; (2) no documented HIV transmission to dental workers has yet been reported; and (3) 23 out of 31 documented transmissions and 22 out of 65 possible transmissions have occurred in either nurses or in clinical laboratory workers. Both of these groups commonly work with needles and draw blood specimens from patients. It is also well documented that blood is the most common body fluid present in occupational transmission, and most reported health-care injuries involve needlesticks or other sharps accidents. With specific reference to dental professionals, six have been investigated and are without identifiable behavioral or transfusion risks. In each instance, percutaneous or mucocutaneous occupational exposures to blood or other body fluids containing HIV was reported, but HIV seroconversion was not documented. Even though these figures suggest a very small HIV risk for dental-care providers, we should not interpret them as indicating no risk. Our routine aseptic, barrier, sterilization, and disinfection infection-control precautions have gone a long way in establishing a high standard of practice safety for both health-care workers and those receiving care. These should not be compromise, be-

[advertisement omitted]

cause HIV does not seem to be a major occupational infection for dental workers.

My thoughts on the questions surrounding mandatory HIV testing for health-care workers and notification of infection status to patients are based on guidelines that I was pleased to help develop for the Michigan State Department of Public Health.[2] The original recommendations of our task force, which were published in 1991, specifically addressed HIV; however, the scope was expanded in 1992 to include hepatitis B-virus-infected health-care workers.

Central to the debate of HIV-infected health-care workers is: What is considered an acceptable level of risk of HIV transmission? The CDC has periodically updated retrospective studies of patients of HIV-infected health-care workers. At present, HIV test results obtained from more than 18,000 of the patients of 32 HIV-infected health-care workers demonstrate *no* HIV infections that could be attributed to medical or dental care, except for the dental patients in Florida. To place these risks in better perspective, they should be compared to other risks patients face as they enter the health-care system. For example: *1 out of every 10,000* persons undergoing general anesthesia dies; *1 to 2 out of every 100,000* persons treated with penicillin have an adverse (anaphylactic) reaction resulting in death.[2]

Some elected officials would like to pass legislation mandating HIV testing for health-care workers, with those found to be infected being required to disclose their HIV status to patients. Many of us who work in the area of infectious diseases see substantial problems

Table 1--Health-Care Workers with HIV Infection and/or AIDS, With Documented and Possible Occupationally Acquired HIV Infection, by Occupation, Reported Through June 1992[1]

Occupation	Documented Occupational Transmission Number	Possible Occupational Transmission Number
Dental workers, including dentist	0	6
Embalmer/morgue technician	0	3
Emergency medical technician/paramedic	0	7

Health aide/attendant	1	5
Housekeeper/maintenance worker	1	5
Laboratory technician, clinical	11	10
Laboratory technician, nonclinical	1	1
Nurse	12	12
Physician, nonsurgical	4	7
Physician, surgical	0	2
Respiratory therapist	1	1
Surgical technician	0	1
Technician/therapist, other	0	3
Other health-care occupations	0	2
TOTAL	31	65

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[Add. 64]

with this line of thinking. Many health-care workers who know they have personal risk factors for HIV infection would be tested anonymously, or would avoid testing altogether. Thus, the regulations would only succeed in driving the problems underground. Neither mandatory testing nor mandatory disclosure is justified given the extremely low risk of HIV transmission from HIV-infected health-care workers to patients.

Because the risk of HIV transmission from patient to health-care worker is much higher than the risk of transmission from health-care worker to patient, mandatory testing of health-care workers is also not a defensible position without a mandate for routine testing of patients. Because of the window period for development of positive serological reactions following HIV infection, retesting of subjects to determine their infection status would have to be done on a regular basis. In effect, many millions of scarce health-care dollars would have to be diverted from effective HIV prevention strategies to finance such testing.

It is also unfortunate that the intent of proposed mandatory testing and disclosure policies appears to be aimed at automatically limiting the practice of HIV-infected health-care workers. I believe that, in addition to the factors mentioned above, other negative consequences of such policies would occur. These include: (1) the loss of services of noninfected health-care workers who choose to modify their practice or change careers based on fears of occupational seroconversion and its consequences; (2) the loss of

employment of HIV-infected health-care workers including their valuable services to society; (3) the probable reduction in services for HIV-infected patients, including denial of care by health-care workers who fear seroconversion; and (4) the negative (social, psychological, and financial) impact of false-positive tests resulting from widespread testing in populations with a low incidence of disease. Some argue that patients have the right to know whether or not their health-care workers are HIV-infected. Patients have the right to any information that might be reasonably expected to affect their health status. In general, however, the HIV infection status of health-care workers does not meet this criterion because of the extremely low risk of HIV transmission from the infected health-care worker, and the standard of infection control routinely practiced during patient treatment. What reasonable and scientific actions do we take, then, instead of mandatory testing and patient notification? Stay tuned.

[References omitted]
[Advertisement omitted]

[Add 65]

[Ms. Abbott's Motion for Summary Judgment page 17]

***

On November 7, 1994, after Sidney Abbott had filed a complaint against Dr. Bragdon with the Maine Human Rights Commission, Dr. Bragdon -- for the first time -- applied for hospital privileges at Down East Community Hospital ("Down East") in Machias, Maine. See R.A., Ex. R.[22] Down East twice requested additional information from Dr. Bragdon. R.A., Ex. T. Dr. Bragdon did not provide the requested information. Dr. Bragdon does not today have privileges at Down East or any other hospital.

[Notes 20 and 21 omitted]

[22] Machias is approximately a two hour drive from Bangor. In addition, the cost of the hospital operatory at Down East is five dollars per minute. See transcript of deposition of David Kennedy, Account Officer at Down East, R.A., Ex. S: 5. Assuming even a thirty minute procedure, this would increase the cost of filing a cavity from $35 to $45 to $185 to $195. The cost of the operatory could be greater if the head nurse who is in charge of billing determined that the hospital should bill for the time it takes Dr. Bragdon to set up his equipment and supplies. R.A., Ex. S:10.

[Add. 66]

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A. We use single gloves when we do examinations.

Q. Okay. Did Judith Bragdon ask you anything else?

A. No.

Q. Did you have any conversations with anyone else in your office on September 16th, 1994, about Sidney Abbott prior to meeting Sidney Abbott?

A. No.

Q. Tell me what happened when Sidney Abbott entered your treatment room. What was the first thing you said to her?

A. I can't remember. Probably small talk. Usually I comment on the weather or something like that, but I can't remember.

Q. Prior to Sidney Abbott's entering your treatment room, had you determined in your own mind whether you would provide dental treatment to her or not?

A. I didn't know what she wanted done.

Q. Prior to Sidney Abbott's entering your treatment room -- well, by dental treatment, how do you define dental treatment?

A. I don't understand the question.

Q. Okay. Let me step back and rephrase that. Prior to entering -- Sidney Abbott entering your treatment room, had you made any determination about whether you would provide a service to her

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such as filling a cavity?

A. Not specific to her, no.

Q. Okay. Prior to meeting Sidney Abbott, did her HIV status -- her HIV-positive status -- had you made any decision about what you would and wouldn't provide to her on September 16th, 1994, on the basis of her HIV-positive status?

MR. GILBERT: Are you focusing the witness's attention on the approximately one-minute time frame that he talked about earlier between when he reviewed the form and when he showed the patient into the office, in that one minute?

MR. KLEIN: Right.

Q. Prior to meeting Sidney Abbott.

A. No.

Q. Okay. Describe to me everything you can remember about what Sidney Abbott said to you and what you said to her.

A. I think I already have.

Q. Okay. Is there anything else?

A. Not that I can recollect right now.

Q. Okay. What -- did you take -- tell me what type of dental examination or procedures you performed

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on Sidney Abbott?

A. She said -- I examined the lesion which she was inquiring about, and then I did a general exam.

Q. Okay. And what does that consist of specifically?

A. Basically, it's a general look at the soft tissue and a general look at the other teeth to see if there is any major problems.

Q. Okay. What instrument --

A. With the mirror and explorer.

Q. I'm sorry?

A. A mirror and an explorer.

Q. Tell me exactly what you did with a mirror and explore?

A. What do you mean?

Q. How did you use them to perform the dental exam?

A. Took the mirror in my left hand and the explorer in my right hand and examined the teeth. I may have used the mirror to retract her cheek, too. I sometimes do during an exam.

Q. And you noticed on the health registration form that Ms. Abbott indicated that she had a toothache?

A. Yes.

Q. And did you take any steps to determine whether

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Ms. Abbott's toothache was indicative of something other than a cavity such as the -- such as the dental conditions which you described earlier that can be -- that a toothache would be indicative of?

A. No, because I did ask her if it was bothering her, and she said no. She said that she had been recently examined by another dentist.

MR. KLEIN: Can we have this as the next exhibit?

(Bragdon Exhibit #14 marked.)

BY MR. KLEIN:

Q. Can you identify Exhibit 14?

A. Yes. This is the record of Sidney Abbott with the front and back page.

Q. Can you please read into the record all of the notations that you placed on that record?

A. "9/16/94, buckle decay, number 18. HIV diagnosed eight years ago. Wants to know cost. 35 to 45 dollars. Would have to do in hospital outpatient because of infectious disease policy. Will think about and get back to us." And it's noted under the charges, no charge.

Q. If Sidney Abbott had not indicated on the Patient Registration and Health Record that she is HIV

[Add. 67]

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Q. Describe to me the circumstances under which you would have filled Sidney Abbott's cavity.

A. If she had indicated that she wanted to have it done, I would have made arrangements, if possible to do it in a hospital setting.

Q. What hospital?

A. I'm not sure.

Q. Why aren't you sure?

A. Because I've never had to take and make arrangements for such a thing.

Q. What hospital would have attempted to treat Sidney Abbott in?

A. Probably Downeast Memorial Hospital.

Q. Where is that?

A. In Machias. Did I say Downeast Community Hospital?

Q. That's in Machias?

A. Yes.

Q. How many miles is Machias from Bangor?

A. I'm not sure.

Q. Do you have an estimate?

A. Probably 60 miles or thereabouts.

Q. Are there any other hospitals aside from Machias that you would have considered treating Sidney Abbott in?

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A. I would have considered Eastern Maine or St. Joseph's Hospital.

Q. Any others?

A. No.

Q. Where is St. Joseph's Hospital located?

A. In Bangor.

Q. As of September 16th, 1994, you didn't have privileges at -- to use the facilities of any hospital, is that correct?

A. That is correct.

Q. And prior to September 16th, 1944, had you ever inquired about obtaining privileges at any hospital?

A. No.

Q. And had you ever even inquired about the process for obtaining privileges at any hospital prior to September 16th, 1994?

A. No.

Q. Okay. At some time subsequent to September 16th, 1994, did you have any communication with the Eastern Maine Medical Center?

A. No.

Q. Okay. At some time subsequent to September 16th, 1994 -- strike that. On September 16th, 1994, if Ms. Abbott had wanted you to fill her cavity, was

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Eastern Maine Medical Center the first place that you would have inquired into with respect to using a hospital operatory?

A. Yes.

Q. All right. And at some time after September 16th, 1994, did you learn anything about Eastern Maine Medical Center that would affect your ability to obtain privileges there to use a dental operatory?

A. Yes.

Q. What did you learn?

A. I learned that you were planning to depose Dr. Paul LaMarche.

Q. When did you learn that?

A. From my counsel, John McCarthy.

Q. When did you learn that?

A. I can't remember for sure.

Q. Continue.

A. And that flagged us that there must be some problem at Eastern Maine which you planned to amplify on. So with that I called Dr. Chris Maller.

Q. Can you spell the last name?

A. M-A-L-L-E-R.

Q. Okay. Was that prior to LaMarche's deposition?

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A. Yes.

Q. And who is Dr. Maller?

A. He is a general dentist in Bar Harbor who also has an office up here.

Q. In Bangor?

A. Yes. I knew he did some hospital dentistry, and at the time I thought he was in the Eastern Maine facility. So I was going to inquire about their facilities and application and whatnot. He advised me that the head of dental services at Eastern Maine was also the head of dental services at St. Joseph's Hospital. It was a problem in that he was against general dentists working in a hospital setting.

Q. Who is the head of dental services at Eastern Maine Medical Center that you are referring to?

A. Dr. Daniel McCarthy.

Q. What else did Dr. Maller tell you?

A. He told me that as a result he couldn't make any headway with getting hospital privileges at Eastern Maine, and he understood that they had allowed the dental facility to deteriorate.

Q. Did he tell you specifically how they -- did he tell you specifically or -- did he tell you specifically what kind of dental facility they

[Add. 68]

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had there?

A. No.

Q. Do you know what kind of dental facility they had there?

A. No.

Q. Do you know what he meant by they had allowed the dental facility to deteriorate?

A. He indicated he didn't think you could do general regular dentistry there anymore.

Q. Okay.

A. And he indicated that he was doing his hospital dentistry at the Bar Harbor hospital on administrative privileges permission.

Q. Okay. Did you have any further conversation with Dr. Maller about hospital privileges?

A. No, I didn't.

Q. After your conversation with Dr. Maller, you didn't have any communications with Eastern Maine Medical Center?

A. No.

Q. At any time have you had any communications with the St. Joseph's Hospital about obtaining privileges?

A. No.

Q. Okay. You have never even -- have you ever

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inquired about the process for obtaining privileges at St. Joseph's Hospital?

A. No.

Q. Are there any other hospitals in the Bangor area at which you would have applied for privileges to treat Sidney Abbott?

A. No.

Q. Okay. Did you ask Dr. Maller what you should do to obtain hospital privileges somewhere?

A. In the sense of specific steps, no. I asked him -- he indicated that Dr. McCarthy was just implacable, and he didn't think that anything could be done with him at either hospital from his personal experience; and since we was in litigation, I wasn't too anxious to take and get into an adversarial situation.

Q. With whom?

A. With Dr. McCarthy.

Q. Okay. Your understanding was that the hospital, Eastern Maine Medical Center, did not have the dental facilities you needed, is that right?

A. He indicated that, too. My understanding at the time that Sidney Abbott came in was that they maintained them and general dentistry could be

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done at that facility without any problem.

Q. Okay.

A. Because that had been the state of affairs in previous years.

Q. And you've since learned that that was not correct, is that right?

A. That's right.

Q. All right. That as of September 16th, 1994, Eastern Maine Medical Center had not maintained its dental facilities, is that right?

A. That was my understanding.

Q. Do you know what facilities St. Joseph's Hospital ever had for dental treatment?

A. No.

Q. Okay. Did you ever inquire whether St. Joseph's Hospital ever had the kind of dental equipment you would need to treat Sidney Abbott?

A. No.

Q. Have you ever applied for privileges to use a hospital surgical operatory or any other aspect of a hospital to provide dental services to a patient?

A. Besides BMHI, which we have already discussed, and I have also applied to Downeast Community Hospital in Machias.

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MR. KLEIN: Let's go off the record for a second.

(Off the record colloquy.)

BY MR. KLEIN:

Q. When did you apply for privileges to Downeast Hospital in Machias?

A. I can't remember the exact date. It's dated on the letter. It's on the cover. It's November something.

Q. You think it was in November of 1994?

A. I think so.

Q. Prior to submitting the application to Machias, did you have any written or verbal communications with anyone at Machias?

A. Yes.

Q. Who did you speak with or what was the mode of communication?

A. Telephone.

Q. Who did you call? Did you call someone?

A. Yes.

Q. Who?

A. I called Dr. Charles Dorr.

Q. Can you spell the last name?

A. D-O-R-R.

Q. Who is he?

[Add. 69]

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A. He is a general dentist --

Q. Okay.

A. -- who is head of dental services down there.

Q. How did you get Dr. Dorr's name?

A. I knew him. I knew he was doing hospital dentistry down there.

Q. All right. How did you know that?

A. Previous conversations.

Q. What was your conversation with Dr. Dorr?

A. I asked him about getting courtesy privileges, and his response was: Do you have an HIV patient that you want to treat? And I said I may have. And he indicated he didn't think there would be any problem with getting privileges and sent the form out the next day.

Q. Okay. Did you first tell Dr. Dorr that you may have an HIV-positive patient?

A. No. He asked me.

Q. Okay. So you didn't mention -- he asked you whether you wanted to treat prior to your mentioning anything about having an HIV-positive patient, is that right?

A. Let's back up a bit. I asked him about the hospital privileges, and that was his response

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because he is aware of my infectious disease policy.

Q. All right. Was Dr. Dorr at the time that you called him aware of the litigation in the case known as John Doe versus Randon Bragdon?

A. Yes, he was.

Q. How was he aware of that; do you know?

A. From the mailings.

Q. What mailings?

A. That I had mailed out to the members of the dental society in '91.

Q. Did those mailings reference the lawsuit brought by John Doe?

A. Yes.

Q. Had you prior to this conversation had any --

A. Let me think here a minute. It referenced the proceedings of the Maine Dental Society. It may not have been the lawsuit itself. I believe there had been an article or two in the paper, or three.

Q. Prior to your conversation with Dr. Dorr about obtaining privileges at Machias, had you ever had a discussion with him about your infectious disease policy?

A. No.

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Q. Okay. Had you ever had a discussion with him about the previous lawsuit against you brought by John Doe?

A. No.

Q. What else did you discuss with Dr. Dorr in this conversation?

A. That was it. That wasn't it. I asked him what they had for a facility there, if we would be able to do general dentistry, and he said yes.

Q. Tell me everything you know about the facilities for dentistry at Machias.

A. He said that they had a chair, delivery unit and a hospital operatory for it.

Q. If that everything you would need?

A. It was the best I could find at the time.

Q. Well, is it everything you would need?

A. It's not everything I desire. It's the best we could do. That's why we have worked hard to get three state-of-the-art facilities here in the state because we recognize that what we need isn't available.

Q. What is your understanding of the infection control capabilities of the hospital operatory at Machias?

A. I haven't been down there to actually see it, so

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I would have to inspect it, but my understanding is that, one, we would be able to wash it down completely after use. I would be able to set up so that virtually everything that couldn't be autoclaved could be disposable.

Q. Anything else?

A. And they had a filtered-air system.

Q. What do you mean by a filtered-air system?

A. That the air would be filtered for airborne pathogens.

Q. Okay. Is that everything?

A. That's all I'm sure of or I feel sure of.

Q. Have you ever been to Machias to inspect their facilities?

A. No.

Q. Did you ever make any inquiry about the nature of the infection control capabilities of the facilities at Machias other than this conversation you are relating with Dr. Dorr?

A. No.

Q. If you had privileges to use the facilities at Machias on September 16th, 1994, would you have treated Sidney Abbott in that facility?

A. Yes.

MR. GILBERT: You're assuming for

[Add. 70]

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A. That I am writing for my expert position.

Q. Is there more of your expert position coming to us in the future?

A. No. As documentation for what the position is.

Q. Pardon me?

A. It will be a footnoted documentation of my position.

Q. Okay. So you will be supplementing your expert position with additional information?

A. No.

MR. McCARTHY: That comes from counsel.

MR. KLEIN: Well, what is the story?

MR. McCARTHY: There is no story. He is referring to materials between counsel and --

MR. KLEIN: Between him and you?

MR. McCARTHY: Yes.

MR. KLEIN: We are getting that, too?

MR. McCARTHY: No.

(Recess taken from 3:04 to 3:19.)

MR. KLEIN: The parties have agreed that Dr. Bragdon's deposition will be resumed on Tuesday, June 13th, and Wednesday, June 14th. The parties will agree that Dr. Bragdon's deposition will be continued on the days of June 13th and June 14th from the hours of 9:00 to 6:00

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with an hour for lunch and reasonable breaks and, that with respect to the documents, that the plaintiff is seeking to compel from Dr. Bragdon -- Dr. Bragdon's counsel agrees -- that we are not prejudiced with respect to seeking a further resumption of Dr. Bragdon's deposition if the judge or magistrate judge orders it along with our motion to compel.

MR. McCARTHY: That's correct.

MR. KLEIN: Anything else?

MS. PERLEY: The United States and the Maine Human Rights Commission will be asking questions on the 13th, and counsel for Sidney Abbott will be asking questions on the 14th.

MR. McCARTHY: And Dr. Bragdon doesn't concede that the United States has the right to ask questions, and it's understood that we are not waiving that right by allowing you to ask questions on the 13th.

BY MR. KLEIN:

Q. Dr. Bragdon, you just went through a number of different changes which you are using to support your expert position that universal precautions are not adequate. Is that correct?

A. That's correct.

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Q. Are there any other examples that you care to provide in response to that answer other than what you have just given?

A. No.

Q. Calling your attention again to your expert report which is Exhibit 7, at page two you have listed a number of considerations that you think are necessary for treatment of an HIV-positive patient in a hospital operatory, is that correct?

A. Yes.

Q. All right. With respect to air filtration, what is air filtration? Strike that question. And these considerations for hospital operatory are numbered small Roman number I through XX. Could you please go over each of those and tell me how it creates an advantage over your private dental operatory with respect to infection control?

A. Yes. Air filtration filters the air in the operatory which minimizes airborne pathogens both rising from the procedure and also from the ambient. For example, immunocompromised patients are quite sensitive to aspergillus, aspergiliosis (sic).

Q. Can you spell that?

[Page 336]

A. A-S-P-I-R-G-I-L-L-I-O-S-I-S (sic).

Q. What is that?

A. It's an infection. It's a fungus-type infection HIV patients get quite easily, and it's also a threat to other immune-compromised patients. It doesn't take much to expose the environment to it. For example, lifting out a tile to repair a light or something can put it into the air, and it's part of standard hospital infection control if they are anticipating remodeling to move patients from there to put hepa filters in at the very least. They usually try to put barriers in, too.

Q. Is this something that you are concerned about HIV-positive patients contracting or that you are concerned by HIV-positive patients transmitting to --

A. Contracting.

Q. -- staff?

A. Contracting. Ultraviolet lights. They are another extra measure in killing pathogens. Negative pressure operating room provides protection from airborne pathogens leaving the operatory area.

Q. I'm sorry, I just didn't hear that.

[Add. 71]

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A. It minimizes the chance of airborne pathogens leaving the operatory area to get into other areas outside the operatory. These are used quite commonly even for patient rooms that there is either a known or potential problem with respiratory disease, airborne disease. Respirators, these provide a higher level filtration than the standard ear-loop surgeon's masks we use. Sterile water supply should be

used with three-way syringe and the high-speed hand piece. And the primary pathogens that are threats of that are pseudomonas and legionella bacteria.

Q. If I could just ask one question. What is pseudomonas? And how do you spell that?

A. P-S-E-U-D-O-M-O-N-A-S.

Q. And what is it?

A. If I remember correctly, it's antero bacteria.

Q. It's a what?

A. If I remember correctly, which I may not, it's a gram negative antero bacteria; A-N-T-E-R-O bacteria.

Q. And what is its significance with respect to this case?

A. It causes disease.

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Q. And what about legionella?

A. It causes Legionaire's Disease.

Q. And I'm sorry, continue on with respect to the sterile water supply.

A. That's it.

Q. Is your concern about the sterile water supply -- what is your purpose in using sterile water supply?

A. It's to protect the patient.

Q. From pseudomonas and legionella?

A. There is other pathogens, but those are two primary ones.

Q. What other pathogens are you trying to protect from the patient?

A. I'm not sure it's well documented, but it is documented that in many dental delivery systems that the water has high levels of bacteria; and as you know, even bacteria that normally don't produce disease can in an immune-compromised patient.

Q. Okay.

A. I had another thought that slipped my mind. There has been one case of an orthodontist that they believe died as a result of Legionaire infection from a dental unit.

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Q. Okay. If you can continue with each of the areas.

A. Okay. Hair coverings should be worn to prevent contamination of hair by droplets and splashes.

Q. And if I can just -- as we are going through each one of these, since the list is long, my question is how each one provides an advantage over your dental operatory.

A. With the standard precautions we take, we don't cover our hair.

Q. Okay.

A. A face shield should be worn to protect skin areas not protected by the masks; safety glasses, from droplets and splashes: Double gloving.

Q. How does that provide an advantage over your dental operatory?

A. It doesn't in itself, but this should be done. I'm not saying that every one of these things couldn't be done in the operatory, but I am saying the presentation was to give indication of what types of things should be done.

Q. Okay.

A. Double gloving should be used to minimize penetration of skin -- to skin. Gloves should be changed every 30 minutes for extended procedures

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to minimize leaks and hands washed before regloving. Treatment should be carefully planned so that in as far as possible, all dental materials and items to be used are transferred to disposable containers in appropriate amounts to eliminate contamination of kits. Disposables should be used as much as possible. Shoes should be covered. In all probability hospital scrubs should be worn. These should be changed as procedures have been completed, placed in appropriate container hamper. Second assistant should be present to minimize cross contamination. In as far as possible, direct passing of instruments from dentist to assistants should be eliminated. Hoses and cords should be covered. There is also advantage in the hospital setting in case of medical emergency. Standard hospital post-operative cleanup protocols are adequate. They wash down the floors between procedures and things like that which normally wouldn't be done in a dental operatory. As far as possible, instruments should be cleansed and sterilized at the hospital. Equipment which cannot be heat sterilized . should be thoroughly clean disinfected. Use of such equipment should

[Add. 72]

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be avoided whenever possible. That concludes it.

Q. Okay. Dr. Bragdon, are you able to quantify the risk of HIV transmission in a private dental operatory?

A. Yes.

Q. What is that?

A. The risk of transmission in the dental operatory if you have a percutaneous injury is approximately .5 percent.

Q. what is the risk of HIV transmission in -- are you able to quantify the risk of HIV transmission in a hospital surgical operatory?

A. Yes.

Q. What is that?

A. If you have a percutaneous injury, it's .5 percent.

MR. KLEIN: Let's go off the record.
(Off the record colloquy.)

BY MR. KLEIN:

Q. Dr. Bragdon, what is the -- is the risk of obtaining or sustaining a percutaneous injury in a hospital operatory the same as or different than the risk of sustaining a percutaneous injury in a private dental operatory?

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A. It would be less.

Q. And why is that?

A. Because you take different protocols than what you ordinarily take.

Q. What protocols do you take?

A. What protocols?

Q. With respect to -- which make a lesser risk of sustaining a percutaneous injury in a hospital setting?

A. One, you would have the second assistant so that your chairside assistant could concentrate more on what she was doing, not to have those distractions. Two, we would do what we could to minimize passes between; certainly minimize them as much as possible so that you minimize injuries in making passes. The double gloving will provide some but not a whole lot of protection.

Q. Is that everything?

A. That takes care of the percutaneous-injury aspect.

Q. Are you able to double glove in your private dental operatory?

A. Yes.

Q. Okay.

A. But I have explained before that it is not

[Page 343]

practical on a regular basis.

Q. Are you able to utilize a second assistant in your private dental operatory?

A. No.

Q. And why is that?

A. There is not room.

Q. Okay. In your view, what is the -- are you able to quantify the risk of HIV transmission in a dental operatory from a nonpercutaneous event?

A. No.

Q. Are you able to quantify the risk of HIV transmission in a hospital surgical operatory from a nonpercutaneous event?

A. No.

Q. Do you believe that there is any risk of HIV transmission in a dental operatory from a non percutaneous event?

A. Yes.

Q. Do you believe that there is any risk of HIV transmission in a hospital surgical operatory from a non percutaneous event?

A. Yes.

Q. Are you able to quantify the difference in risk of HIV transmission from a nonpercutaneous event in a hospital surgical operatory as opposed to a

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private dental operatory?

A. I don't think that information is --

Q. I'm asking you if you, as an expert, are able to quantify the difference in risk between -- from a nonpercutaneous -- start again. Can you quantify the difference in risk of HIV transmission between a nonpercutaneous injury in a hospital operatory and a dental operatory?

A. No.

MR. McCARTHY: We need to take just a second.
(Recess taken from 3:57 p.m. to 4:01 p.m.)

BY MR. KLEIN:

Q. We're back on the record.

A. I would like to clarify my answers to the last two questions. You asked if I could quantify the difference and that I can't, but certainly we can infer that there would be a significant improvement between a dental operatory and a hospital operatory.

Q. But as you sit here today, you're not able to quantify or -- you are not able to quantify the difference in risk of HIV transmission from a nonpercutaneous event between a hospital surgical operatory and a dental operatory, is that right?

[Add. 73]

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A. I am able to show that these recommendations which I have talked about before are, in fact, used in a hospital setting to reduce the risk and that with other diseases, that they do reduce the risk of transmissions.

Q. Okay. What diseases are you using to show that a hospital setting reduces the risk of transmission of HIV from a nonpercutaneous event?

MR. GILBERT: Well, I object to the form of the question. I think you may have meant to but you did not explicitly incorporate his assumption that it's not just a physical hospital operatory, but it's one that is using these infection control devices.

MR. KLEIN: Yeah.

MR. GILBERT: If you incorporate that as part of the assumption of your question, then I withdraw my objection.

BY MR. KLEIN:

Q. What other diseases -- for what other diseases is there a risk of reduction due to a nonpercutaneous event using the hospital operatory features that you have outlined at small Roman numerals I through XX on pages 2 and 3 of your expert report, and I mean reduced risk

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of transmission of HIV?

A. Repeat the question.

Q. Let's have the question back.

(Pending question read back.)

MR. KLEIN: I guess I withdraw that question.

Q. Is it your testimony that the infection control features that you have outlined at pages 2 and 3 of your expert report reduce the risk of transmission of diseases other than HIV by nonpercutaneous means?

A. That wasn't what I said.

Q. Okay. What did you say?

A. I said that -- you had originally asked if I could quantify, and I had said no to that; but I wanted to clarify the fact that even though we can't quantify, we can demonstrate there is a reasonable expectation that there would be a significant reduction in the risk.

Q. In the risk of what?

A. Of transmission.

Q. Of what?

A. HIV and other diseases.

Q. Do you know of any study or publication -- strike that. What are you pointing to to demonstrate

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this reduction in risk?

A. What do you mean what am I pointing to?

Q. You say we can demonstrate that there is a reduction in the risk of transmission of HIV and other diseases. What is your evidence for that?

A. Fundamentally, the reason that these hospital operatories are built this way is because it has been demonstrated that these features work.

Q. For what?

A. For reducing the risk of transmission of disease.

Q. With respect to air filtration, how many air exchanges would you -- are, in your opinion, necessary to treat an HIV-positive patient?

A. The recommendation for a room in which air exchange is being engineered in -- for example, TB is six changes an hour.

Q. Do you know whether the operatory at Machias has six changes of air per hour?

A. I don't know.

Q. Okay. Do you use nitrous oxide in your office?

A. No, I don't.

Q. Do you know how many air exchanges you have in your office?

A. I have no idea.

Q. How does the use of air filtration -- does the

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use of air filtration reduce the risk of a percutaneous transmission of HIV?

A. No.

Q. Is it your opinion that the use of air filtration reduces the risk of a nonpercutaneous transmission of HIV?

A. Yes.

Q. How does it do that?

A. It filters out pathogens in the air, and it has been demonstrated that from the aerosols generated from a hand piece burr in vitro that the free virus can infect a culture; and in the area in vivo, it has been demonstrated that there are live virus aerosolized into the air. Nobody has been able to show the significance of them, but it has been demonstrated that they are present.

Q. Okay. Do you know what the significance of them is?

A. The significance is it may be very well possible to be HIV infected from that, although it hasn't been proven yet.

Q. From an aerosol droplet?

A. Yes.

[Add. 74]

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Q. Describe to me how ultraviolet lights reduce either the percutaneous or nonpercutaneous transmission of HIV.

A. They provide a kill of pathogens in the air and also on surfaces that are irradiated with ultraviolet.

Q. Are you able to use ultraviolet lights in your office?

A. No.

Q. Why is that?

A. Not setup for it.

Q. How do you have to be set up for it?

A. You would have to have special engineering.

Q. Is it your position that ultraviolet lights would kill aerosolized HIV?

A. It's my position that they would help. I don't think there is any studies showing the exact efficacy of them.

Q. What do they do then?

A. I just said what they did.

Q. They kill pathogens?

A. Yes.

Q. In the air?

A. Yes.

Q. And on surfaces?

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A. And on surfaces.

Q. How does the negative pressure operating room affect the percutaneous or nonpercutaneous transmission of HIV?

A. The negative pressure takes and prevents pathogens that are airborne from leaving the room; and TB, as I said before, is a high-incidence disease. Kaposi's sarcoma is believed to be of the human herpes.

Q. It's a herpes?

A. It's believed to be caused by one of the human herpes viruses.

Q. Is it a herpes virus?

A. It's of the human herpes class. It's not herpes one or two, and there is a research paper that indicates that they don't feel that those types of infections can be prevented from transmission in the regular dental operatory.

Q. What types of infections?

A. Pardon?

Q. What type of infection are you talking about?

A. Human herpes and Kaposi's sarcoma is one of them, and approximately 10 percent of AIDS patients have Kaposi's sarcoma infection.

Q. All right. Are you able to use a respirator in

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your private office?

A. Not on a regular basis, no.

Q. Why not?

A. I'm not equipped for it, and they are difficult to breathe with on a regular basis.

Q. What about on an occasional basis?

A. It wouldn't be very cost effective.

Q. Why?

A. I have never needed one yet.

Q. What is your understanding of how you would use a respirator in the hospital operatory?

A. You would use it in place of the regular surgeon's mask.

Q. What does it look like? Strike that. Have you ever seen a respirator that you're talking about here?

A. Yes.

Q. What does it look like?

A. It takes and covers the nose and this area here with a seal, and the ones that are preferable have removable, replaceable filter cartridges.

Q. And it's your testimony that you could not use such a respirator in your private office?

A. It would be very impractical, yes.

Q. Why?

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A. One, it's expensive and, two, it would be difficult to use on a regular basis.

Q. Why is it difficult to use? I'm just trying to understand that.

A. They are difficult to breathe through.

Q. Do you know how much they cost?

A. No, I don't.

Q. What would you have to do to obtain -- strike that. Does a sterile water supply reduce the risk of transmission of HIV in a hospital surgical operatory?

A. you should take and disinfect the lines afterwards because it has been documented that the lines have been infected with HIV following work on an HIV patient.

Q. That the lines have been infected with HIV?

A. Yes.

Q. Does a respirator reduce the risk of transmission of HIV?

A. Yes.

Q. How?

A. Because it minimizes the risk of breathing in aerosolized droplets of HIV-contaminated blood.

Q. Are you able to use a sterile water supply in your office?

[Add. 75]

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A. Yes.

Q. How does a hair covering reduce the risk of HIV transmission, if at all?

A. It protects the hair from splashes.

Q. Are you able to use a hair covering in your private dental office?

A. Yes.

MR. KLEIN: Can we take a five-minute break?

MR. McCARTHY: Sure.
(Recess taken from 4:18 to 4:20 p.m.)

BY MR. KLEIN:

Q. In your expert opinion, does a face shield reduce the risk of transmission of HIV?

A. Yes, it does.

Q. Okay. And how is that?

A. It would protect areas that weren't covered.

Q. Can you wear a face shield in your office?

A. Yes.

Q. In your view, does -- in your expert opinion, does double gloving reduce the risk of HIV transmission?

A. If you minimize passes, then it would reduce the risk, some, yes.

Q. Well, just does double gloving itself reduce the

[Page 354]

risk of HIV transmission?

A. It's a complicated question because it's -- as we said in the previous deposition, I have tried double gloving, but the loss of tactile sense prevents using it on a routine basis. So with the lack of tactile sense, you increase your chances of percutaneous injury unless you have done something else to minimize that chance; and if you have done that, then there would be a net improvement. If you haven't, there wouldn't be.

Q. Okay. Are you able to wear two pairs of gloves in your office?

A. Yes.

Q. Does changing gloves every 30 minutes for extended procedures reduce the risk of HIV transmission?

A. It certainly does.

Q. How so?

A. Because the gloves develop defects in them in addition to the ones you start out with. Over a period of time, the longer the period of time, the more defects. In addition to that, your hands start to sweat. The wetter your hands are inside the glove, the more transmissions that you have through pathogens that do get through the

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glove.

Q. Can you change your gloves every 30 minutes in your office?

A. Yes.

Q. Does carefully planning treatment as far as possible -- I'm sorry. Does carefully planning treatment so that all dental materials and items to be used are transferred to disposable containers in appropriate amounts to eliminate contamination of kits, does that reduce the risk of HIV transmission?

A. Yes, it does.

Q. Can you carefully plan treatment so that all dental materials and items to be used are transferred to disposable containers in appropriate amounts to eliminate contamination of kits in your office?

A. It can be difficult.

Q. Why is it?

A. Because you would have to tear the whole office apart.

Q. How so?

A. Because we are set up with all of these things around and virtually all of the available space is used. You would have to take and move

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everything out, and we really don't have any place to move it to.

Q. But you could do it?

A. It would be very impractical.

Q. When you anticipate treating a patient in a hospital surgical operatory, do you know whether you could transfer all of your dental materials in advance to disposable containers to eliminate contamination of kits? Do you know whether you could do that in the hospital surgical operatory?

A. Yes.

Q. How do you know that?

A. What do you mean how do I know that?

Q. Well, have you ever seen a hospital -- well, strike that. When was the last time you saw a hospital dental operatory?

A. It has been a while.

Q. How long?

A. Maybe 10, 12 years.

Q. Okay. And have you ever seen the hospital dental operatory at Machias?

A. No.

Q. And have you ever inquired about their facilities or equipment?

A. Yes. I have inquired about their equipment.

[Add. 76]

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Q. What have you asked them?

A. I asked them what they had.

Q. What did they tell you?

A. They told me they had a patient chair and the delivery system.

Q. What is a delivery system?

A. That's the system by which you take and have your hoses, hand piece attachments, the three-way syringe, opportunity to place your medicaments and other materials you need.

Q. What else do they have?

A. I'm not sure. I haven't been down there. They indicated there wouldn't be any problem doing it. They had what was needed to do it.

Q. The actual dental materials that you would use in treating someone at Machias, do you bring those?

A. Yes.

Q. You bring those yourself?

A. Yes.

Q. Where do you get them from?

A. I would make them up from the kits we have.

Q. Okay. How would you do that?

A. What do you mean how would I do that?

Q. Describe to me the process of -- let's say you're going to take equipment to fill someone's cavity

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to Machias. Tell me how you go about gathering your equipment, what you would need to bring for dental materials?

A. Depending on what we was planning on doing, we would take and -- some materials already are in cupules. We would plan on which cupules we needed so that we would just have those with us and the appropriate amount. Some materials are in tubs, and things like that could be loaded into cupules --

Q. What are those?

A. Cupules. They are a small container that you can put small amounts of different items into.

Q. Okay.

A. And in some cases, with planning you could use the end of a bottle or something like that with an appropriate amount for the planned treatment.

Q. All right. Would those be disposable containers?

A. Yes.

Q. So you would put everything that you need into disposable containers?

A. They would be disposable in the sense that you would use a container that you could dispose of when you got done with it.

Q. And then you would take those to Machias?

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A. Yes.

Q. All right. By the way, do you know if Machias has a sterile water supply in their dental operatory?

A. I don't know.

Q. Can HIV be transmitted through hair?

MR. GILBERT: Through what?

MR. KLEIN: Hair.

A. Are you saying hair or air?

Q. Hair.

A. It could in the sense that if you had a droplet of blood which got on the hair and then you ran your hands through your hair later. Say you came out of the operatory and you had degloved and taken your mask off and you had a cut on your hand that it touched, you could very well get it from your hair in that sense. The contact with the hair, the hair being infected, no, unless you squeezed it down into your scalp and a bug had bit you or you scratched it or scratched your head like you're doing now; and you could scratch it into your scalp, too.

Q. You indicate in number 11 that disposables should be used as much as possible in a hospital operatory?

A. Yes.

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Q. Does that reduce the risk of HIV transmission?

A. Yes, it does.

Q. Can you use disposables in your private office?

A. Yes.

Q. Shoes. It says shoes should be covered in number 11. What's the theory there as to the reduction of the risk of HIV transmission by covering shoes?

A. If you get blood splashed on your shoes, it's virtually impossible to disinfect them.

Q. Okay. Can you cover your shoes in your dental office?

A. Yes.

Q. Does the use of -- tell me if I already asked this, but does the use of disposables reduce the risk of HIV transmission?

A. Yes, it does.

Q. It says in number 13 hospital scrubs would be worn and changed after procedures?

A. It says in probability.

Q. What does that mean?

A. It means I haven't confirmed it, but most hospitals require you to use their scrubs.

Q. Would you insist on using scrubs with an HIV-positive patient in a hospital surgical

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operatory?

A. It wouldn't make any difference. We could bring our own clothing or appropriate --

Q. All right. And that's the same clothing -- is that the same clothing that you wear on a daily basis in a private dental office?

A. Yes.

Q. Okay. Now, with respect to an additional assistant, can you describe to me what your normal -- is the purpose of using the second assistant to eliminate the risk of an injury in passing instruments?

A. And also to minimize cross contamination.

Q. Okay. And how does it eliminate cross contamination?

A. Because everything -- it's similar to the circulating nurse on a regular surgical team and so that the surgical team never leaves the operating site. The circulating nurse hands them everything so that -- and she is not contaminating it.

Q. But is it true that the purpose -- the second assistant is there to participate in the passing of dental instruments?

A. No.

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Q. Why is she there? What does the second assistant do?

A. She's there to assist in passing, preparing some things to minimize cross contamination.

Q. Okay. Do you ever pass instruments -- in the hospital operatory, would you pass instruments at all back and forth between you and your second assistant?

A. No.

Q. Would you pass instruments back and forth between yourself and your regular assistant?

A. As little as possible.

Q. But would there be some passing of instruments?

A. It depends on the procedure.

Q. Let's say it's filling a cavity.

A. I don't know.

Q. Why don't you know?

A. I haven't sat down and worked it out.

Q. Okay. So in terms of working with your regular assistant and your second assistant, would you have to devise a new plan to determine how you were going to physically access your dental instruments?

A. Yes.

Q. Have you devised that plan?

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A. No.

Q. Okay. Do you have any idea today what that plan would be?

A. Yes.

Q. Before we get to what it is, is it a plan that you would ever use in your private dental operatory with your regular assistant?

A. No.

Q. Would you describe to me what the plan is?

A. The plan would be to arrange the instruments in such a way that they could be placed, and I would take them myself in hand and place them back; and when she needed to move them, they would be fixed. This is a common technique that is used with HIV patients in surgery. With surgery patients, they have a magnetic holder. For example, if there is room across the torso or the legs, the instruments will go on there and be placed there, and then the nurse will take them from there rather than being handed back and forth.

Q. And that's all done with your second assistant?

A. No. The first assistant.

Q. Okay. Do you know who your second assistant would be?

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A. Probably my wife.

Q. Has she ever been trained in this procedure?

A. No.

Q. How often does your wife serve as your regular assistant?

A. Probably several times a week.

Q. Several times a week?

A. Yes.

Q. But not on a full-time basis?

A. Not usually.

MR. KLEIN: Okay. Let's go off the record.
(Off the record colloquy and the deposition concluded at 4:32 p.m.)

(All exhibits retained by Mr. Klein.)
(The right to read and sign was exercised.)

[Add. 78]

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want to anesthetize the lower left side the teeth, then I would take and turn my left hand upside down, insert the thumb into the notch of the ascending ramus and then grasp the back of the ramus or back of the jaw with this finger.

Q. This finger being the index finger?

A. The index finger, and with the other fingers may or may not stabilize them against the head, depending upon the position and the size of the patient's head, and then take the syringe and insert it beside the thumb at a trajectory calculated to impact about two millimeters in front of where this nerve bundle and the artery and the vein enter, and then I aspirate to make sure that we weren't into a vein or an artery and then inject most of the copula there, sometimes all of it, depending on the particular procedure, and then withdraw, and I might inject about a tenth of a copula near the surface to be sure we get the lingual nerve.

Q. If you were treating a patient who was HIV positive, would you do that any differently?

A. Probably try to stabilize the head more than I would ordinarily.

Q. And how would you do that?

A. Be sure to place these fingers --

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Q. These fingers being your pinky, ring finger and middle finger?

A. Yes, and also I could position my body up against the head too.

Q. And when administering an infiltration would you do anything differently in administering the anesthetic?

A. Yes.

Q. What would you do then?

A. Stabilize the head to take extra precautions.

Q. Okay. Okay. In the year 1994 how many needlesticks did you receive?

A. I don't know.

Q. Can you approximate?

A. Probably in the vicinity of about six or so.

Q. And in any of those instances did you draw blood?

A. Yes.

Q. How many?

A. All of them.

Q. Did they occur while you were administering an anesthetic or in another situation?

A. Those occurred in administering anesthetic.

Q. Have you ever received a needlestick other than while administering anesthetic?

A. I have stuck myself suturing.

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A. Anything else?

A. No.

Q. You said that you received approximately six needlesticks in 1994. Would you say that was true, again approximately, for the years 1990 to 1994?

A. Yes.

Q. Okay. And again, from 1990 have you ever received a needlestick other than while administering anesthetic or while suturing a patient?

A. No.

Q. Has any member of your staff ever received a needlestick?

A. No.

Q. Has any member of your staff ever received a sharp's injury?

A. Yes.

Q. How many times has that occurred?

A. Twice.

Q. Can you tell me about those two times?

A. My assistant, Tracy Frost, was preparing instruments to be cycled through the chemclave, which is our heat sterilization unit, and in blotting the instruments she drove an explorer tip through her finger, her middle finger.

Q. When you say through her middle finger, did it go

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directly through from one side to the other?

A. No, it didn't.

Q. You had said the subcutaneous injury before. Was that the right phrase?

A. It went into the tissue below the skin, yes.

Q. What about the other time that there was a sharp's injury?

A. I think it was a few months later she did the same thing.

Q. Anything else?

A. Not that I know of.

Q. Other than the needlesticks you just described, have you ever received any other type of sharp's injury while practicing dentistry?

A. Yes.

Q. Tell me about those.

A. I get sharp's injuries from explorers, knives, burs, sharp edges on partials, in particular, although occasionally a sharp edge on a denture, too. Amalgam saw.

MR. McCARTHY: I didn't hear that.

A. Amalgam saw. Sharp pieces of tooth and bone, matrix bands.

Q. I'm sorry, what?

A. A Matrix bands. There's probably other things, but

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that's what comes to mind at the time.

Q. Approximately how many sharp's injuries of the type that you just described would you say you receive in a given year?

A. Probably several a week. Last week I had two in one day.

Q. And do these all occur while you are treating a patient?

A. Those all can occur while I am treating a patient, yes, and those two last week that occurred on the same day occurred while treating a patient, different patients.

Q. And have you drawn blood in all of these injuries?

A. It's hard to quantify it. Most of them, yes.

Q. Have you taken any steps to reduce either the number of needlesticks that you have had occur or the number of sharp's injuries that have occurred?

A. Yes.

Q. What steps are those?

A. To try to pass the instruments so that the -- well, I try harder to protect my assistant and the patient than I do myself, because you can't achieve an optimum for everybody, so in consideration of that, I try to protect the patient and my assistant the most.

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Q. Have you taken any steps to reduce the number of needlesticks or sharp's injuries that you have received?

A. Yes.

Q. And what are those steps?

A. One is that we, in as far as possible, don't handle the syringe until the patient is ready for the injection, try not to startle them, try not to inject when they don't seem like they might necessarily be cooperative. We -- the biggest single thing, and I think the reason that I do have a good record with this is that we stop everything when we are handling a syringe, nothing else is done, and nothing else is done until the needle is capped and the syringe laid down.

Q. How do you cap your needles?

A. One hand scoot.

Q. The sharp's injuries that you were mentioning just before from the explorers, the knives, the burs, the amalgam saw and the like, do those mostly occur from -- while passing instruments or while you are working on the patient?

A. Mostly while working on the patient.

Q. Is that because your hand is in the patient's mouth?

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A. Mostly, yes.

Q. Is there anything about working in a hospital dental operatory as compared to your office that reduces or minimizes the number of needlesticks and other sharp injuries that you receive?

A. Yes.

Q. What is that?

A. We double glove, and as far as possible we would eliminate passes. We evaluate the patient as to whether he could control his mouth and body. That's a hypothetical question, so it's hard to be hypothetical.

Q. That's fine. Could you double glove in your office for a particular patient?

A. Yes.

Q. Could you eliminate passes as much as possible in your office?

A. It would be difficult.

Q. Why?

A. Because we are set up for a chairside assistant.

Q. And why couldn't you change your setup?

A. Because the office operatory is designed that way. It would be difficult to have the assistant and the second assistant as I think would be desirable and to position the cart optimally, too.

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Q. How would you ideally eliminate passes as much as possible in a hospital operatory? Could you describe what you would do to eliminate the passes? Where would you have the cart set up?

A. It would depend on what type of delivery system they had. If they had an over-the-patient delivery system, you could

probably use that to place instruments and then take the instruments off from that.

Q. And that, if I am correct, would be a table or something of that sort that would actually be placed over the patient, him or herself?

A. Yes.

Q. Okay. If they had a different type of delivery system, where would the cart be placed?

A. If they had a side delivery system, the optimum position would be for it to be beside the patient where you could reach with the right hand for instruments, or other items are placed in convenient order and reach.

Q. What type of delivery system do you have in your office?

A. Side delivery.

Q. And could you place the side delivery beside the patient so that you could reach with your right

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Q. Are you making any representations to your patients about the safety of receiving treatment in your office?

A. No.

Q. If a patient says to you is it safe for me to be treated in your office, what is your response?

A. My response is that I am doing the best I know how with the information I have available and the instruments and other modalities that are available.

Q. Doing the best you know how with respect to what?

A. To the manner in which I am practicing.

Q. To make it safe, to what?

A. To provide treatment and to make it safe.

Q. You said just now that hepatitis B is a model for HIV?

A. Yes, I did.

Q. What do you mean by that?

A. That hepatitis B has been used as a model for HIV.

Q. In what respect?

A. To model its modes of transmission.

Q. How else?

A. To model the efficacy of various products.

Q. Is it your position that to the extent a precaution is ineffective in preventing the transmission of

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hepatitis B it is equally ineffective in preventing the transmission of HIV?

A. It's my position that hepatitis B has been given as a model for HIV and its modes of transmission have been stated to be the same by the CDC and others, and from our

experience with hepatitis B we know that the present precautions that are being taken are ineffective.

MS. PERLEY: Can you read back my question, please?

(Preceding question read back.)

Q. I don't believe your last answer answered that question.

A. I believe it did.

Q. So you're saying -- I want to make sure I am right. To the extent a precaution is ineffective with respect to the transmission of hepatitis B, it is equally ineffective with respect to the transmission of HIV. Is that your position?

A. That's the model that's been given.

Q. And do you agree with that model?

A. I think it's the only model we have to work with right now.

Q. So again, notwithstanding the model, because the model for the mode of transmission may be different

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than the efficacy of the precaution, and my question is to the extent a precaution is ineffective or has been demonstrated to be ineffective in preventing the transmission of hepatitis B, is it your position that it is equally ineffective in preventing the transmission of HIV?

A. My position is there's not enough information to answer that question absolutely.

Q. Okay.

A. But from what they've presented for using it as a model, it would be reasonable to assume that the efficacy would be similar.

Q. I'm going to shift gears a little bit. You said before that you're licensed to practice general dentistry in the State of Maine. What are the prerequisites to being licensed to practice dentistry in the State of Maine?

A. You have to be a graduate of an American dental school, you have to pass the written national dental boards, and you have to take and pass the clinical northeast regional dental boards.

Q. Anything else?

A. I can't remember exact details. You have to be of good character, no felony convictions, things like that, and then the Maine Board of Dental Examiners,

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taking these things all into account, would decide whether or not to grant a license to practice in the State of Maine.

Q. Are there any grounds on which you can lose your license?

A. Yes.

Q. What are those?

A. Malpractice.

Q. Anything else?

A. I believe some felony convictions, I'm not sure just how the dividing line goes. You can also lose your license for drug and alcohol abuse.

Q. Anything else?

A. You could lose your license if you were shown to be physically or mentally impaired in such a way that you could not perform -- be reasonably expected to perform competent dentistry.

Q. Anything else?

A. There may be others, but that's what comes to mind now.

Q. Have you read the Maine Dental Practice Act?

A. Yes, I have.

[Add. 81]

Defendant's Exhibit 6

Down East
Community Hospital

R.R. 1 BOX 11 MACHIAS, MAINE 04654 (207) 255-3356

March 31, 1995

Randon E. Bragdon, DMD
30 Judson Boulevard
Bangor, Maine 04401

Good Morning Dr. Bragdon:

In order to further process your application for appointment to the Medical Staff of our Hospital, we are seeking some additional information, as follows:

1. Please provide further details regarding the so-called John Doe case which has been discussed in various press accounts, including sharing with us:

   a) Copies of any court pleadings;

   b) Copies of any correspondence to or from the Maine Human Rights Commission;

   c) Copies of any documents reflecting the disposition of the matter by the Maine Human Rights Commission;

   d) Further detail from you as to how this suit relates to your "longstanding infectious disease policy" and your planned response to this litigation.

2. Please provide further details regarding the lawsuit brought in early December in U.S. District Court by Sidney Abbott, including the following:

   a) Copies of any court pleadings;

   b) Copies of any correspondence to or from the Maine Human Rights commission;

   c) Copies of any documents reflecting the disposition of the matter by the Maine Human Rights Commission;

   d) A further statement from you as to the nature of this litigation and your planned response.

3. Please share with us the nature of your "longstanding infectious disease policy".

[Add. 82]

4. If your policy is to refuse to treat AIDS and HIV patients in your office, how do you respond to statements in the press from a spokesman for the Mine[sic] Dental Association, Frances Miliano, that the "refusal to treat (in an office) based solely on the patient's HIV-positive status is unethical" based on policy statements from the ADA?

5. Please review the enclosed statement from the ADA regarding its view of ethical duties for dentists in terms of treating HIV positive patients in the dentists' offices and indicate whether you follow this view, and if not, why not.

6. Are you under any review by the State Board which licenses Dentists?

7. Are you still a member in good standing of the Maine Dental Association or under any review by that body?

8. Is it your intention to treat patients at our Hospital who might otherwise be treated at your or another dentist's office, and if so, is this cost-effective treatment for these patients?

9. Please provide any other detail you would care to share with this Hospital regarding your policy, these lawsuits, and the controversy surrounding the matter.

The Hospital's interest is in reviewing the nature of care you provide, or decline to provide, against applicable ethical and legal standards governing your profession. It is further interested in evaluating whether it is cost-effective to treat patients in a hospital setting who might otherwise be treated in a dentist's office.

We look forward to your response to each of the preceding questions in order to evaluate your application. We will not deem your application complete until we have these materials.

Sincerely,

George Avery, FACHE
Administrator

p.c.: F. James Whelan, M.D., Chairman, Credentials Committee

[Add. 83]

AIDS and the mouth

Diagnosis and management of oral lesions

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Munksgaard

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AIDS and the mouth

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[Add. 85]

[Omit first three paragraphs]

Among the most dangerous risks are probably needlestick injuries and puncture injuries with sharp instruments, the use of which abounds in dentistry. The number and type of exposures vary from specialty to specialty. These include exposures to blood, sharp bone fragments, wires, sharp scalers, reamers, files, bands and pliers. Dentists administer large numbers of local anesthetic injections and current designs of local anesthetic syringes oblige the practitioner to recap the needle frequently. The CDC recommend that needles should not be recapped, bent or broken by hand. The ADA suggest that if needles are left uncapped they should be placed in a separate working area to avoid injury, or the needles may be recapped if the hand is protected from injury by using a device or holder. There are many devices available to the dentist to aid in recapping, and one method is to use the scoop method where the cap is left on the work surface and the needle is returned to the cap in a scoop without opposing the hand to the needle (Figs. 18.6-8). In 1987, an ADA survey reported that general dentists gave an average of 177 local

[Add. 86]

anesthetic injections per month and that the rate for needlestick injuries was 1.5 per 100 injections.[2]

All instruments that are used clinically should be sterilized. This is best achieved by the use of heat including autoclaving, hot air sterilization or chemical vapor sterilization. After clinical use, instruments should be cleaned before sterilization. This can be done by scrubbing or by the use of an ultrasonic cleaner. Handpieces should ideally be autoclaved although some alternatives are discussed in the CDC guidelines (see below).

***

[Add. 87]

15 June 1993 Volume 118 Number 12

Annals of Internal Medicine

Surveillance of HIV Infection and Zidovudine Use Among Health Care Workers and Occupational Exposure to HIV-infected Blood

Jerome I. Tokars, MD, MPH; Ruthanne Marcus, MPH; David H. Culver, PhD; Charles A. Schable, MS; Penny S. McKibben; Claudia I. Bandea, PhD; and David M. Bell, MD, for the CDC Cooperative Needlestick Surveillance Group*

■ *Objective*: To study the risk for human immunodeficiency virus (HIV) infection and the patterns of use and associated toxicity of zidovudine among health care workers after an occupational exposure to HIV.

■ *Design*: An ongoing, prospective surveillance project conducted by the Centers for Disease Control and Prevention.

■ *Participants*: Exposed workers voluntarily reported by 312 U.S. health care facilities from August 1983 to June 1992.

■ *Results*: Four of 1103 enrolled workers with percutaneous exposure to HIV-infected blood seroconverted (HIV seroconversion rate 0.36%; upper limit of the 95% C1, 0.83%); no enrolled workers with mucous membrane (n = 75) or skin (n = 67) contact seroconverted. During October 1988 to June 1992, 31% of 848 enrolled workers used zidovudine after exposure; this proportion increased from 5% during October through December 1988 to 43% during January through June 1992. Despite using zidovudine after exposure, one worker became infected with a strain of HIV that was apparently sensitive to zidovudine. Adverse symptoms, most commonly nausea, malaise or fatigue, and headache, were reported by 75% of workers using zidovudine; 31% of workers did not complete planned courses of zidovudine because of adverse events.

■ *Conclusions:* The risk for HIV seroconversion after percutaneous exposure to HIV-infected blood is 0.36% which is similar to previous estimates. Zidovudine is used after exposure by a sizable proportion of health care workers enrolled in the project despite frequent, minor, associated symptoms. Documented failures of postexposure zidovudine prophylaxis, including in one worker enrolled in this study, indicate that if zidovudine is protective, any protection afforded is not absolute. Postexposure zidovudine, if used, requires careful consideration of possible risks and benefits.

Annals of Internal Medicine 1993:118:913-919

From the Hospital Infections Program and the Division of HIV/AIDS. National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia. For current author addresses, see end of text.

For members of the surveillance group, see the Appendix.

Health care workers are at risk for human immunodeficiency virus (HIV) infection after an occupational exposure to blood, certain other body fluids, and tissue from an HIV-infected patient (1, 2). Zidovudine has been used after exposure to reduce this risk, despite the lack of data on efficacy and limited data on toxicity when used for this purpose (3-5). In 1983, the Centers for Disease Control and prevention (CDC) began a national voluntary surveillance project to estimate the risk for HIV transmission after a single exposure to HIV-infected blood (6). In October 1988, the project was expanded to collect information describing the use of postexposure zidovudine among enrolled workers. In this update of the ongoing surveillance project, we focus on the patterns of use and associated toxicity of postexposure zidovudine use among enrolled workers and report the failure of zidovudine to prevent HIV infection in one worker.

Methods

CDC Surveillance Project

The surveillance project has been described previously (6, 7). In brief, workers in a group of participating health care institutions throughout the United States are voluntarily enrolled by cooperating

investigators after an exposure to blood from a patient with documented HIV infection as a result of percutaneous injury (for example, a needlestick or a cut from a sharp object), contamination of mucous membranes, or contamination of nonintact skin. (Extensive or prolonged blood contact with intact skin may constitute an occupational exposure [4], but workers with intact skin exposures are not enrolled in this project.) Epidemiologic information and a blood specimen for HIV serologic testing are collected by the cooperating investigator at a baseline visit within 1 month after exposure and at follow-up visits 6 weeks, 3 months, 6 months, and 1 year after exposure. At enrollment, workers complete and mail directly to the CDC an anonymous questionnaire that includes information on nonoccupational risk factors for HIV infection. Workers who seroconvert are interviewed in person by an investigator using a standard CDC protocol for the interview of persons with no identified risk.

Human immunodeficiency virus serologic testing is done either at the CDC or at the cooperating institution's laboratory, with all positive results confirmed at the CDC.

Use of Postexposure Zidovudine

In October 1988, the surveillance project was expanded to collect additional information on the postexposure use and

toxicity of antiviral agents, such as zidovudine. A standardized protocol for offering or using postexposure zidovudine was not provided by the CDC; in accordance with the Public Health Service statement (4), individual cooperating investigators and exposed workers determined whether postexposure zidovudine would be used, and, among those using zidovudine, the dosage and duration of prophylaxis.

Adverse Events

At the 6-week follow-up visit, the cooperating investigators were asked to record reported symptoms on a standardized checklist provided by the CDC, as well as the zidovudine regimen, if used. In this surveillance project, a standard protocol for laboratory testing for adverse effects (for example, anemia) of zidovudine was not provided by the CDC; such tests were done at the discretion of the cooperating investigator, who recorded the test date and any results outside the testing laboratory's normal range.

Statistical Analysis

Data were analyzed using PRODAS (Conceptual Software, Houston, Texas). Epi-Info (CDC, Atlanta, Georgia), and the Statistical Analysis System (SAS Institute, Inc., Cary, North Carolina). The upper bounds of the 95% CI were calculated using the binomial distribution. Because of missing data, totals for specific analyses may not equal the total number of enrolled workers.

Results

From August 1983 through June 1992, 1245 workers from 312 institutions were enrolled and tested for HIV antibody at baseline and at least 180 days after exposure. These workers comprised nurses (64%), physicians and dentists (12%), phlebotomists (8%), laboratory workers (6%), medical students (2%), housekeepers (1%), and other workers (such as technicians and physician and nursing assistants) (8%).

Exposures to HIV-Infected Blood and Risk for HIV Infection

A total of 1245 workers were enrolled and tested for HIV antibody at baseline and at least 180 days after exposure. These workers had been exposed to blood from source patients who had AIDS as defined by the CDC surveillance case definition in use at the time of enrollment, (1012 [81%] exposures) (8), who were HIV-antibody positive and symptomatic but who did not have AIDS (68 [5%] exposures), or were HIV-antibody positive and asymptomatic (165 [13%] exposures). Exposure types were percutaneous injury (1103; 89%) and blood contact with mucous membranes (67; 5%) or non-intact skin (75; 6%). The 1103 percutaneous injuries were caused by syringe needles (375; 34%), intravenous needles (343; 31%) suture needles (41; 4%) and other needles (228; 21%); scalpels (41; 4%); lancets (27; 2%); and other sharp objects (48; 4%).

Among 1103 workers with percutaneous injuries who were HIV seronegative at baseline, 4 (0.36%; upper limit CI, 0.83%) seroconverted to HIV. Among workers with percutaneous injury, the seroconversion rate after exposure to blood from source patients with AIDS was 0.44% (4 of 899; upper limit CI, 1.01%); the seroconversion rate was not statistically different after exposure to

blood from source patients who were HIV-antibody positive and symptomatic but who were without AIDS (0 of 57 $P > 0.2$) or source patients who were HIV-antibody positive and asymptomatic (0 of 147, $P > 0.2$). No seroconversions occurred among workers with blood exposure of mucous membranes (67 workers; upper limit CI, 4.31%) or skin (75 workers; upper limit CI, 3.87%).

An additional 497 workers enrolled in the early 1980s, before HIV-antibody testing was available, were tested for HIV antibody at least 180 days after exposure but not at baseline. One of these workers was HIV-antibody positive when first tested 10 months after exposure (7). At that time, a sex partner of this worker was also tested and found to be HIV seropositive; the dates of HIV infection for this worker and partner are unknown. Including these 497 workers, the HIV seroprevalence rate was 0.32% (5 of 1532; upper limit CI, 0.68%) after percutaneous exposure, 0% (0 of 100; upper limit CI, 2.9%) after mucous membrane exposure, and 0% of 110; upper limit CI, 2.7%) after skin exposure to HIV-infected blood.

Of the four workers enrolled in this project who have seroconverted after exposure to HIV, three have been previously described (6, 7). The fourth worker, a female laboratory worker, was injured in 1992 by a 21 gauge syringe needle after doing a phlebotomy on a patient who was known to have AIDS. The worker reported no behavioral risk factors for HIV infection, except for having had sexual contact with a person at risk for HIV infection more than 1 year before her injury. The worker began taking zidovudine 2 hours after the injury, in a dose of 100 mg. five times a day, for the first day and then 200 mg. five times per day for 16 additional days (Table 1, report 5). The worker discontinued use of zidovudine after 17 days because of nausea, fatigue, and myalgia. Thirty-eight days after exposure, the worker developed fever, malaise, fatigue, nausea, arthralgia, myalgia, and rash. Serologic test results for HIV were negative for specimens of the workers blood collected on the day of the injury and 6 weeks afterward, were indeterminate by enzyme immunoassay and positive by Western blot 3 months after injury, and were positive by both enzyme immunoassay and Western blot 4 months after injury.

The source patient was not known to have received zidovudine before the needlestick incident.

To determine whether the worker was infected with zidovudine-resistant virus, peripheral blood mononuclear cells were collected from the worker and the source patient 3 months and 8 months after exposure, respectively. At the Walter Reed Army Institute of Research (Washington, D.C.), cells from the worker were positive for HIV-1 using the polymerase chain reaction (PCR), but efforts to isolate the virus for phenotypic zidovudine-susceptibility testing were not successful. Nested PCR to detect mutations in the HIV reverse transcriptase at amino acid position 215 associated with zidovudine resistance did not show alterations at that site in peripheral blood mononuclear cells from either worker or source patient (9) (D. Mayers, Personal communication). At the CDC, direct sequencing of the HIV-1 reverse-transcriptase gene (amplified by PCR from the worker's peripheral blood mononuclear cells [10]) showed no mutations at position 215 or at the four

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Table 1. Reported Instances of Failure of Postexposure Zidovudine To Prevent HIV Infection in Health Care Workers after Percutaneous Exposure to HIV-Infected Blood*

Report	Country	Year	Sharp Object	Hours to First Dose	Regimen† mg Days	Onset of Retro-viral Illness	Time Seroconversion Documented‡	Source Patient on Zidov-udine	Refe-rence
1	South Africa	1992	IV cannula	0.5	1200 42	None	6 wk	No	(29)
2	United States	1991	22-gauge phlebo-tomy needle	0.75	800 10	2 wk	3 mo	Yes	§
3	A Western European country	1992	18- to 20-gauge IV cannula	1	1000 42	2 wk	56 d	Yes	‖
4	France	1990	Phleboto my needle	1.5	1000 21	16 d	52 d	Yes	(30)
5	United States	1992	21-gauge syringe needle	2	1000 17	38 d	121 d	No	This report

6	United States	1990	16-gauge IV cannula	3 to 7	1000	8	36 d	94 d	Yes	§
7	Australia	1990	Hollow needle	6	1000	54	5 wk	6 wk	Yes	(31)
8	South Africa	1990	Lancet	12	1200	21	17 d	24 d¶	no	(29)

*HIV = human immunodeficiency virus; IV = intravenous.
† Regimens are expressed as milligrams of zidovudine per day and number of days taken. Report 5: 500 mg/d for 1 day, then 1000 mg/d for 16 additional days.
∏ By enzyme immunoassay and Western blot.
§ C. Ciesielski, unpublished CDC data.
ll Confidential communication from the country's national public health authority to D. Bell, CDC; cited with permission; and also **Anonymous: HIV** seroconversion after occupational exposure despite early prophylactic zidovudine therapy. Lancet. 1993; 341-1077-8.
¶ At 24 days, reactive by enzyme immunoassay and weekly positive by Western blot. AT 3 months, enzyme immunoassay had a higher optical density reading, and Western blot had strongly positive bands.

other amino acid positions (positions 41, 67, 70, and 219) known to be associated with zidovudine resistance (11, 12) (data not shown).

Use of Postexposure Zidovudine

From October 1988 to June 1992, the period when use of zidovudine was studied, 848 workers were enrolled. Postexposure zidovudine was used by 265 (31%) of these workers. Of 200 cooperating investigators who enrolled workers during this period, 110 (55%) reported that at least one worker used zidovudine. Zidovudine was prescribed in doses ranging from 200 to 1800 mg/d (median, 1000 mg/d) and for periods of 1 to 180 days (median 42 days). The interval from exposure to first dose of zidovudine ranged from less than 5 minutes to 17 days (median, 4 hours). No clinically significant changes in prescribed regimens of zidovudine were seen during the study period (data not shown).

The proportion of enrolled workers using zidovudine increased from 5% in the fourth quarter of 1988 to 50% in the third quarter of 1990 and has been stable subsequently, averaging 43% during January to June 1992 (Figure 1). Physicians, dentists, and medical students were more likely to use zidovudine than other health professionals (59 to 135 [44%] compared with 206 of 713 [29%], $P = 0.001$), as were workers with percutaneous injury

compared with contamination of mucous membranes or skin (244 of 750 [33%] compared with 21 of 98 [21%], $P = 0.03$).

Among 219 workers for whom information was available, the source of payment for zidovudine was the employer (69%), workers compensation (17%), private insurance (8%), the workers themselves (2%), and other or multiple sources (9%). Sources of payment for office visits and laboratory studies were similar. Median charges, excluding the cost of the time spent by hospital employee health care and infection control personnel, were reported for some workers who took zidovudine (n = 60 workers), $132 for office visits (n = 31 workers), and $132 for laboratory testing (n = 41 workers).

Adverse Events

Three workers who planned to use zidovudine for only 1 day were excluded from the tabulation of adverse events. Information on symptoms that occurred during the 6 weeks after HIV exposure was available for 236 (89%) workers using zidovudine; 176 (75%) reported one or more symptoms, most commonly nausea, malaise or fatigue, or headache (Table 2). Symptoms were reported less frequently among workers who did not use zidovudine (26% of 439 workers, $P < 0.01$; Table 2). Cough or acute respiratory illness was the most common symptom among those not using zidovudine.

Limited information was available on hemoglobin or hematocrit values of workers using zidovudine. Of 175 workers who completed 21 or more days of prophylaxis 51 (29%) had paired hemograms at least 21 days apart; among these 51 workers, 7 (14%) had a 10% or greater reduction in hemoglobin or hematocrit values.

One or more symptoms occurred in a similar proportion of workers taking less than 1000 mg per day (75%

[Add. 90]

[Figure 1 omitted]

of 56 workers), 1000 mg per day (73% of 105), and more than 1000 mg per day (77% of 73). Seventy-four (31%) workers did not complete their planned regimen of zidovudine because of adverse symptoms (73 workers) or reduction in hemoglobin level (1 worker). Of these 74 workers, 43 (58%) stopped taking zidovudine, 14 (19%) used a lower dose, and 13 (18%) used a lower dose and later stopped taking zidovudine (information missing on 4 workers). For an additional 15 workers, zidovudine was discontinued to reduced in dosage for reasons not related to toxicity (for example, the workers changed their minds). Twenty-eight (12%) workers were absent from work for periods ranging from 1 to 49 days (median, 2 days) because of adverse events attributed to zidovudine.

Discussion

Based on data from this ongoing surveillance project, the risk for HIV-infected blood is 0.36%, similar to earlier reports from this and other studies (2, 6, 13). Although mucocutaneous blood contact has resulted in HIV transmission (14, 15), none of the workers enrolled in this project with mucous membrane or skin exposures tested positive for HIV. One worker enrolled in this project seroconverted despite taking zidovudine for 17 days after exposure; however, the number of enrolled workers is insufficient to assess zidovudine efficacy.

Failure of Postexposure Zidovudine Prophylaxis

Including the worker described in this report, the CDC is aware of eight reported instances of failure of postexposure zidovudine prophylaxis after percutaneous injuries to health care workers (*see* Table 1). In these cases, zidovudine was begun 30 minutes to 12 hours (median, 1.75 hours) after exposure and was used in doses of 800 to 1200 mg/d (median, 1000 mg/d) for 8 to 54 days (median, 21 days). The workers in reports 4 and 8 (*see* Table 1) stopped zidovudine because of evidence that they were seroconverting; for the other six workers, it is uncertain whether

longer periods of prophylaxis would have been more effective. An additional five instances of failure of postexposure zidovudine have been reported after exposures in which the quantity of HIV-infected blood injected was larger than would be expected from a needlestick. These five include two instances of accidental intravenous inoculation of blood or other material from patients with HIV infection during nuclear medicine procedures (16, 17). They also include one instance each of a blood transfusion (4), suicidal self-inoculation (18), and an assault on a prison guard with a needle syringe (19). In most instances, information is not available on whether the source patient was taking zidovudine, which might have led to development of a zidovudine-resistant strain of HIV. In our case, the source patient was not known to have been taking zidovudine before the needlestick exposure, and no zidovudine-resistant HIV strains were detected by genetic sequencing or PCR techniques. These data suggest that the worker became infected with a zidovudine-sensitive strain of HIV, despite taking zidovudine after exposure. Collectively, these case reports indicate that if zidovudine is protective, any protection afforded is not absolute.

Efficacy of Postexposure Zidovudine prophylaxis

Little additional information exists with which to assess the efficacy of postexposure zidovudine. Animal studies using murine and feline retroviruses are encouraging but of limited value because these viruses have pathogenic mechanisms different from HIV (4). Zidovu-

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Table 2. Symptoms Reported by Health Care Workers within 6 Weeks after HIV Exposure, October 1988 to June 1992*

Symptom	Using Zidovudine (n = 236)	Not Using Zidovudine (n = 439)	*P* Value
	n(%)		
One or more symptoms	176 (75)	116 (26)	<0.01

Nausea	17 (50)	13 (3)	<0.01
Malaise or fatigue	79 (33)	31 (7)	<0.01
Headache	59 (25)	13 (3)	<0.01
Vomiting	25 (11)	8 (2)	<0.01
Myalgia or arthralgia	23 (10)	25 (6)	0.07
Abdominal pain or dyspepsia	19 (8)	4 (1)	<0.01
Diarrhea	18 (8)	7 (2)	<0.01
Anorexia	11 (5)	1 (<1)	<0.01
Cough or acute respiratory illness	13 (6)	53 (12)	0.01
Sleep disturbance	8 (3)	0 (0)	<0.01
Fever	10 (4)	20(5)	>0.2
Anxiety	7 (3)	2 (< 1)	0.01
Mental dullness	8 (3)	0 (0)	<0.01
Dizziness	7 (3)	2 (< 1)	0.01
Parasthesia	4 (2)	2 (< 1)	0.2
Sweating	4 (2)	1 (< 1)	0.05
Weight loss	4 (2)	6 (1)	>0.2
Rash	3 (1)	7 (2)	>0.2
Taste disturbance	3 (1)	0 (0)	0.04
Depression	2 (1)	1 (< 1)	0.03
Lymphadenopathy	1 (< 1)	14 (3)	0.04
Asthma	0 (0)	4 (1)	>0.2
Other	17 (7)	26 (6)	>0.2

*HIV = human immunodeficiency virus. Information on symptoms occurring during the 6 weeks after HIV exposure was available for 236 (89%) workers using zidovudine and for 439 (75%) workers not using zidovudine.

dine did not prevent simian immunodeficiency virus infection in monkeys injected with moderate or high inocula or with a rapidly lethal variant of simian immunodeficiency virus (20-22). When lower challenge doses of simian immunodeficiency virus were used and zidovudine was begun before exposure, two of two monkeys in one study and one of six monkeys in the highest initial-dose treatment group in another study did not become infected (23, 24). In a study in SCID-humice (immunodeficient mice transplanted with human hematolymphoid cells), HIV infection was suppressed during drug use, but infection was detected in all animals after discontinuation of zidovudine (25). Because the mice were exposed to HIV using intrathymic injection of a sizable virus inoculum, the relevance of this experiment to needlestick exposure in health care workers is unclear. In a subsequent study (26) in SCID-humice, HIV infection suppression was most effective when zidovudine treatment was begun within 2 hours after exposure, but

the investigators did not determine whether infection was present after zidovudine was discontinued.

Evaluation of the efficacy of postexposure zidovudine in humans is difficult because the HIV seroconversion rate after an occupational exposure necessitates a large sample size to show a prophylactic effect. For example, calculations based on a seroconversion rate of 0.36% after exposure, 75% drug efficacy, 80% power, and a 0.05% one-tailed significance level indicate that more than 2101 workers using postexposure zidovudine would be required to evaluate efficacy. The Burroughs-Wellcome Company (the manufacturer of zidovudine) sponsored a double-blind, placebo-controlled trial to evaluate postexposure zidovudine. The trial was terminated in June 1989, when it became apparent that an insufficient number of exposed workers could be recruited for a randomized trial (27). Because of uncertainty about efficacy and safety, the Public Health Service concluded in January 1990 that a recommendation for or against the use of postexposure zidovudine could not be made (4).

In this surveillance project, the proportion of health care workers using zidovudine increased from 5% in the last quarter of 1988 to 40% to 50% after the January 1990 Public Health Service statement on postexposure zidovudine use, and was more common among physicians, dentists, and medical students than among other workers. The proportion of exposed workers who used postexposure zidovudine was 75% for the University of California San Francisco-San Francisco General Hospital (UCSF-SFGH) and 76% for the National Institutes of Health Clinical Center (NIH-CC) (28). Rates of postexposure zidovudine use may differ among studies because of differences in the types of exposures studied, in the institutional policies about zidovudine availability, and in the knowledge as well as opinions of those who counsel exposed workers.

In this study, 75% of workers using zidovudine reported symptoms, and 31% did not complete their planned regimen because of adverse events. Although only limited data were available to assess the hematologic effects of postexposure zidovudine, no severe sequelae of postexposure zidovudine were

reported. Similar results have been reported by other researchers. In an Italian multicenter study (5), adverse effects (most commonly nausea, asthenia, and vomiting) were reported in 53%, and postexposure zidovudine was discontinued in 25%. In studies at UCSF-SFGH and NIH-CC, 'substantial side effects' were reported by 33% and 24% of workers, respectively; the drug was discontinued in 17% and 50% of workers, respectively (28). In the only placebo-controlled trial (the Burroughts-Wellcome study), nausea, vomiting, and arthralgia were reported more commonly among workers taking zidovudine than among those taking placebo (27).

Limitations of this surveillance project include the possibility that workers or exposures voluntarily reported by workers and cooperating institutions may not be representative of all occupational exposures in the United States. Assessment of short-term adverse effects of zidovudine is complicated by the lack of a standardized postexposure regimen and a placebo control group; also, ascertainment of symptoms may have been more active among zidovudine users than nonusers. Assessment of long-term toxicity is not feasible because of the 1-year duration of follow-up. Detailed information on the nature and frequency of the toxicity of postexposure zidovudine will be determined optimally from carefully supervised clinical trials in selected institutions. The strength of surveillance data include descriptions of the current experience of various hospitals nationwide as

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well as enhanced ability to monitor trends and detect rare events.

Continued assessment is needed of the risk for infection after occupational exposures to HIV and of the risks and benefits of postexposure use of antiviral agents. Additional information about participation in the CDC surveillance project can be obtained from the Hospital Infections Program, National Center for Infectious Diseases, CDC, Mail Stop A-07, Atlanta, Georgia 30333; telephone (404) 639-1547).

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Appendix

The CDC Cooperative Needlestick Surveillance Group consists of cooperating investigators from 312 institutions nationwide. The following investigators enrolled more than 20 health care workers who participated in this study:

[Rest of Appendix and References omitted]

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Cover Story

Percutaneous Injuries in Practicing Dentists

A PROSPECTIVE STUDY USING A 20-DAY DIARY

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ABSTRACT

A novel diary study of 2,304 practicing U.S. dentists examined the frequency and nature of percutaneous injuries over one dentist-month. Burs were responsible for most extraoral injuries (40 percent), and syringe needles accounted for most intraoral injuries (32 percent). The annualized mean injury rate was 3.35, which represents about a threefold decrease since 1987.

The occurrence of percutaneous injuries in the dental office has always been of some concern to dental health care providers, but it traditionally has been relegated to a category of occupational safety issues, such as being injured by an improperly secured tank of gas. However, this issue assumed some importance with the advent of AIDS in 1981 and a higher level of importance when the disease's routes of transmission had been elucidated clearly. Hepatitis B had earlier been identified as a bloodborne infection with potentially very serious consequences, and by the mid-1970s it was known to be at a much higher prevalence in dentists than in the general population.[1]

Subsequently, the AIDS epidemic focused attention on the potential for disease transmission to dental health care providers who are regularly exposed to patients' blood and who are at some risk of percutaneous injuries from needles, burs and sharp and rotating instruments. The concern centered on the risk of transmission, first between infected patient and provider.[2-4] and later between infected provider and patient.[5]

Growing concern in the medical and dental world with the incidence of work-related injuries led to a number of investigations designed to assess the actual risks of transmission. A surveillance study had revealed that the likelihood of health care workers' becoming infected as a result of a percutaneous injury with HIV-contaminated blood was 0.42 percent,[6] but it was not known how often they sustained percutaneous injuries. Researchers undertook a number of studies using a variety of techniques to learn about the incidence of percutaneous injuries; retrospective surveys of injuries incurred during various procedures and using various devices[7]; surveillance studies of health workers with AIDS who had sustained occupational injuries[8,9]; observational studies conducted in operating rooms[10] and hospital-based dental clinics[11]; and a study of medical and

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[Table 1 omitted]

dental equipment that was designed to facilitate greater safety.[12]

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Cover Story

Percutaneous Exposures to HIV-Infected Blood

AMONG DENTAL WORKERS ENROLLED IN THE CDC NEEDLESTICK STUDY

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ABSTRACT

The authors found that 19 percutaneous exposures among dental workers occurred both during and after use of instruments such as syringe needles and scalers. Specific information about the device and action associated with an exposure is important for prevention efforts, including safer instruments and work practices. Most of these exposures probably involved smaller, rather than larger, amounts of blood infected with the human immunodeficiency virus. To our knowledge, none of the exposures resulted in HIV transmission to an enrolled dental worker.

Since August 1988, cooperating investigators from more than 300 institutions such as hospitals, clinics and state and local health departments, have enrolled health-care workers (on a volunteer basis) who have been exposed to blood infected with the human immunodeficiency virus in a national surveillance project conducted by the U.S. Centers for Disease Control and Prevention, commonly known as the CDC. Needlestick Study.[1,2] The primary objective of this national study has been to estimate the risk to HCWs of being infected with HIV after occupational exposure to HIV-infected blood. Using data provided by this project and other studies, researchers have estimated the risk of HIV infection after

percutaneous exposure to HIV-infected blood to be approximately 0.3 percent and have established that the type of occupational exposure most likely to transmit a bloodborne pathogen such as HIV is a percutaneous exposure to blood.[1-4]

This report describes the circumstances of percutaneous exposures to HIV-infected blood that occurred among a small number of dental workers enrolled in this national project. Other studies of occupational exposures among dental workers in the United States indicate that dentists, on average, experience about three percutaneous injuries per year.[5,6] In addition, these studies--which were primarily of dentists and dental students--have described factors associated with injuries, including the types of devices and procedures used and the anatomic site of the injury.[5-10] In most of these reports, however, information has been limited in regard to the action resulting in the injury and the factors likely to be associated with the risk of HIV transmission, such as the depth of the injury or the visible presence of a patient's blood on the instrument. As more data on the specific circumstances of these exposures become available, we will be better able to characterize injuries among dental work-

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TABLE 1

PERCUTANEOUS EXPOSURES AMONG DENTAL WORKERS BY DEVICE (N=19)

DEVICE	NO. OF EXPOSURES
Hollow-bore needles	5
Scaler	4
Explorer	3
Bur	2
Suture needle	1
Surgical wire	1
Periodontal probe	1
Scalpel	1
Other sharp instruments	1

Total	**19**

TABLE 2

PERCUTANEOUS EXPOSURES AMONG DENTAL WORKERS DURING USE OF DEVICE (N=8) BY ACTION

DEVICE	ACTION
Scaler (n=2)	Scaling teeth (n=2)
Suture needle	Tying suture
Surgical wire	Wiring fracture
Hollow-bore needle	Patient grabbed dentist's hand
Explorer	Examining teeth
Scalpel	Manipulating specimen
Other sharp instrument	Not described (dental laboratory)

ers and develop future prevention strategies, such as safer devices and work practices.[11]

Methods

HCWs who sustain a percutaneous, mucous membrane or non-intact skin (for example, chapped or abraded) exposure to HIV-infected blood are eligible to participate in the surveillance project. The HIV serostatus of the exposed HCW is determined at baseline; six weeks after exposure; and three, six and 12 months after exposure. At baseline, the cooperating investigator interviews the exposed HCW to obtain information on the specific circumstances of the exposure.

As of December 31, 1994, cooperating investigators found more than 300 institutions had enrolled 2,860 HCWs in the national surveillance project. Of these HCWs, 19 were dental workers. All enrolled dental workers sustained a percutaneous exposure to HIV-infected blood. We reviewed reports of percutaneous exposures among the enrolled dental workers and examined the circumstances of the exposures.

RESULTS

Of the 19 dental workers reported to have sustained a percutaneous exposure, 10 were dentists (including four dental residents and one graduate student), three were hygienists and six were assistants. Fourteen of these 19 dental workers were based in hospitals. Descriptive information about the severity of the injury was available for 16 wound sites. Fourteen wounds were reportedly of moderate depth with visible penetrations of skin and bleeding. Two were superficial scratches without visible bleeding.

Table 1 lists the devices associated with percutaneous exposures among the dental workers. Hollow-bore syringe needles caused five injuries, three by needles (27 and 30 gauge) on non-disposable aspirating syringes and two by needles (19 gauge) on disposable syringes. Scalers caused four injuries; explorers, three injuries; and burs, two injuries. Six instruments, including three scalers, were reported to have been visibly contaminated with the patient's blood at the time of the exposure.

Exposures were associated with a variety of actions (Figure). Of 19 injuries, eight occurred during use of the instrument or device and 11 occurred afterward. Four of the 11 injuries that occurred after use resulted from picking up sharp instruments during cleanup procedures, either by the handful or in sterilization bags from which contaminated sharp instruments may have protruded. None of these workers wore

utility gloves (reusable and heavy duty) during cleanup procedures. Two injuries occurred during disassembly of the device, one when the worker, while changing a bur in one handpiece, was injured by a contaminated bur in an adjacent handpiece. The other injury occurred when a dental worker attempted to remove a bent, uncapped needle from an anesthetic syringe by hand. Two injuries occurred when workers used a two-handed technique to recap

needles on non-disposable syringes. Injuries in the "other/unknown" category (Figure) included one in which a disposable syringe, dropped by the worker, penetrated the worker's thigh.

Table 2 lists the actions associated with the eight injuries that occurred during use of a device. According to one report, while the worker was removing calculus, the scaler slipped of the tooth and punctured the worker's thumb, which was retracting the patient's lower lip. One injury occurred when a suture needle twisted into the worker's finger while the worker was tying suture thread, and another occurred when a patient suddenly grabbed the dentist's hand during injection of local anesthetic. Presumably, the dentist was injured on the hand that was either retracting tissue or guiding placement of the needle.

The amount of blood from the source patient, as well as the titer of infectious agent in the source blood, may be among the factors associated with the risk of HIV transmission after a percutaneous exposure to HIV-infected blood.

Sixteen exposures were self-inflicted. In only one instance did a co-worker hold the sharp instrument. Sixteen injuries involved the dental worker's hands or fingers. Of these 16 workers, 12 reportedly were wearing gloves and at least three were wearing two pairs of gloves. At least 12 of the 19 workers knew that the source patient was infected with HIV at the time of the exposure.

Twelve of the exposed dental workers underwent HIV-antibody tests at least six months after the exposure; the results of all tests were negative. Six dental workers were tested soon after they were exposed, and the results of all tests were negative; however, they were not retested six months or later after the exposure. HIV-antibody test results were not available for one worker.

DISCUSSION

The amount of blood from the source patient, as well as the titer of infectious agent in the source blood, may be among the factors associated with the risk of HIV transmission after a percutaneous exposure to HIV-infected blood. In a case-control study that evaluated risk factors for HIV transmission after percutaneous exposures to HIV-infected blood, Cardo and colleagues observed that HCWs were more likely to become infected if they were exposed to a larger quantity of blood and if the source patients were terminally ill with AIDS.[12] In that case-control study, a larg-

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er quantity of blood transferred was represented by the use of large-gauge, hollow-bore needles (<18 gauge), deep injury and the presence of visible blood on the device. In addition, laboratory studies have suggested that during an exposure, less blood is transferred by a needle that passes through a glove, is of smaller gauge or is solid rather than hollow bore.[13]

Six of the devices in the 19 exposures (32 percent) were reported to have been visibly contaminated with the source blood at the time of the injury.

Our findings, including the frequency of visible blood on the device before the injury and the depth of the injury, suggest that, overall, these dental workers may have been exposed to smaller rather than larger quantities of blood from the source patient. Six of the devices in the 19 exposures (32 percent) were reported to have been visibly contaminated with source blood at the time of the injury. Although one 19-gauge needle was among the five hollow-bore needles associated with injuries, most hollow-bore needles used in dentistry to administer local anesthetics are smaller (for example, a 25-gauge needle). Finally, 75 percent of the

workers with hand injuries were wearing gloves at the time of the exposure, and none of the exposures for which information was available was described as involving deep puncture wounds.

Descriptive data about factors such as the amount of source-patient blood to which the worker was exposed can be important as the risk of HIV transmission after percutaneous exposure is considered. Available information indicates that the overall risk of transmitting HIV to workers in dental settings is very small. Through December 1994 no dental workers were among 42 HCWs in the United States who had been documented as having been infected after a specific occupational exposure to HIV.[14] Six dental workers were among the 91 HIV-infected HCWs who possibly had acquired the infection occupationally. For these workers, no other risk for HIV infection could be identified, and each worker reported having been exposed to blood or body fluids in the dental setting. HIV seroprevalence studies also have shown very low rates of HIV infection among dental workers.[15-18]

Our study was limited by the relatively small number of exposed dental workers who participated in this surveillance project and the fact that these dental workers may not be representative of hospital-based or non-hospital-based dental workers. Nonetheless, devices associated with these[19] exposures were similar to those described in several other studies of dental workers. For example, reported percutaneous exposures among students and in some studies, staff at dental schools in the United States commonly were associated with needles (33 to 36 percent), burs (8 to 11 percent) and other sharp instruments (for example, laboratory instruments, scalers and explorers) ($\geq$ 50 percent).[7-9]

We also examined actions associated with the 19 exposures in our study. Previous reports[6,7,10,19] have specified most actions associated with exposures that occur after use of instruments or devices. These actions include handling equipment during cleanup; needle recapping; disassembling a device, such as a bur in a handpiece; or inadvertently touching one instrument (for example, an explorer or bur) when reaching for another instrument.

Researchers have described few specific actions or activities associated with injuries that occur during use of a device (for example, while scaling teeth, injecting anesthetic or wiring a fracture).[6,19,20]

As we examine additional ways to prevent injuries, it can be useful to have detailed information about the circumstances of exposures, including devices and actions. Although some injuries may be difficult to avoid, such as when a sharp instrument or needle is dropped, many injuries may be preventable. Additional efforts could include the use of engineering controls, such as safer devices like self-sheathing hollow-bore needles or dental units designed to shield burs in handpieces placed in the unit. Changes in work practices could include modified techniques that minimize potentially uncontrolled movements of instruments, such as scalers or laboratory knives,[6] under force. Also, dental workers should use instruments instead of fingers to retract or suture tissue in the operative field or guide anesthetic needles during injections. Once developed, preventive interventions must be critically evaluated to determine if the likelihood of injuries among HCWs from needles and sharp instruments is re-

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duced without adversely affecting patient care.[11,21]

Injuries that result from a worker's continued use of unsafe practices, such as two-handed needle recapping, may require additional training or behavioral intervention. Gonzalez and colleagues reviewed exposure reports from one dental school and found that some percutaneous injuries resulted from improper needle recapping and disposal techniques and only sporadic use of heavy-duty gloves during cleanup procedures. Requirements for using heavy-duty gloves during cleanup; needle disposal units in the clinics; and proper recapping methods, such as the one-handed "scoop" technique, were reinforced with students and staff and pertinent information was distributed.[7] Current guidelines state that used needles should never be recapped, purposely bent, broken or

otherwise manipulated with both hands.[21,23] Disposable needles on non-disposable aspirating syringes, however, can be recapped using a one-handed technique or a mechanical device that holds the needle sheath.[22]

Most dental workers in this study reported that they knew the patient's HIV status at the time of the injury. Results of other studies, although limited, have not demonstrated that such knowledge is associated with reduced contact with patient blood from patients, including percutaneous exposures.[24-25]

Injuries that result from a worker's continued use of unsafe practices, such as two-handed needle recapping, may require additional training or behavioral intervention.

CONCLUSIONS

We found that 19 percutaneous exposures among dental workers in our study involved cutting devices, such as scalers and burs; smaller-gauge hollow-bore needles; and explorers. We also found that they occurred during use of a device or afterward during cleanup, disassembly or recapping. Findings representative of the size of the inoculum of blood from the source patient suggest that most of these dental workers were exposed to a smaller, rather than larger, volume of blood. Some injuries could have been prevented if dental workers had adhered to the current recommendations for safe handling of needles and other sharp instruments. Preventing other injuries may require additional interventions, such as the development of safer devices and continued modifications in work practices.

As more is learned about the risk of HIV infection after occupational exposure and the factors associated with that risk, it is important that we continue to assess HCWs, including dental workers, who are exposed to HIV-infected blood. Additional information about participation in the CDC surveillance project can be obtained from the HIV Infections Branch, Hospital Infections Program, National Center for Infectious Diseases, Centers for

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LOW OCCUPATIONAL RISK OF HUMAN IMMUNODEFICIENCY VIRUS INFECTION AMONG DENTAL PROFESSIONALS

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Abstract We studied 1309 dental professionals (1132 dentists, 131 hygienists, and 46 assistants) without behavioral risk factors for the acquired immunodeficiency syndrome (AIDS) to determine their occupational risk for infection with human immunodeficiency virus (HIV). Subjects completed questionnaires on behavior; type, duration, and location of their dental practice; infection-control practices; and estimated numbers of potential occupational exposures to HIV. Serum samples were tested for antibodies to HIV and to hepatitis B surface antigen (unvaccinated subjects).

Fifty-one percent of the subjects practiced in locations where many cases of AIDS have been reported. Seventy-two percent treated patients who had AIDS or were at increased risk for it. Ninety-four percent reported accidental puncturing of the skin with instruments used in treating patients. Adherence to recommended infection-control practices was infrequent. Twenty-one percent of unvaccinated subjects had antibodies to hepatitis B surface antigen. Only one dentist without a history of behavioral risk factors for AIDS had serum antibodies to HIV.

We conclude that despite infrequent compliance with recommended infection-control precautions, frequent occupational exposure to persons at increased risk for HIV infection, and frequent accidental puncturing of the skin with sharp instruments, dental professionals are at low occupational risk for HIV infection. (N Engl J Med 1988; 318:86-90.)

Even before identification of the human immunodeficiency virus (HIV) as the etiologic agent in the acquired

immunodeficiency syndrome (AIDS), the likely routes of transmission were recognized and included homosexual[1-3] or heterosexual[4] sexual activity, exposure to blood or blood products,[5-7] sharing of contaminated injection equipment by intravenous drug abusers,[8] and perinatal transmission from an infected mother to fetus or newborn.[9-11] Since the identification of HIV as the causative agent of AIDS,[12-14] seroepidemiologic studies have supported the idea that these routes are important in transmission.[13-15]

The similarity between the epidemiologic features of AIDS and those of hepatitis B was recognized early in the AIDS epidemic.[16-19] Hepatitis B also occurs with increased frequency among sexually active homosexual men,[20-22] intravenous drug abusers,[22,23] recipients of blood products,[24-27] heterosexual sexual contact of infected persons,[28] and infants born to infected mothers.[29]

Studies in the United States have suggested that dentists are among the health care providers at greatest risk of acquisition of hepatitis B infection.[30,31] The similarity in routes of transmission between hepatitis B and HIV and the recognized occupational risk of hepatitis B infection among dental professionals led us to study the occupational risk for HIV infection among such professionals.

METHODS

From October 1985 through May 1987, dental professionals were recruited for the study by means of a mailing to dentists in the boroughs of Manhattan and the Bronx in New York City, and solicitation at staff and society meetings throughout the New York City metropolitan area and at health-screening activities at the annual meeting of the American Dental Association in Miami Beach in October 1986. Subjects declining participation were asked to

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[Table 1 omitted]

complete (anonymous) a refusal form indicating the reason for nonparticipation. Participants gave written informed consent and completed a questionnaire on demographics: type, duration, and location of practice; behavior placing them at high risk of AIDS; precautions used in treating patients; types and estimated numbers of patients treated; estimated number of accidental parenteral inoculations; and status of vaccination against hepatitis B. The questionnaire had been pretested and modifications had been made to improve the validity of responses. Venipuncture was performed, specimens were coded to ensure confidentiality, and serum was tested for antibodies to HIV by enzyme immunoassay (Abbott Laboratories, North Chicago). Reactive results were confirmed with the Western blot assay.[32] In subjects who had not received the hepatitis B vaccine, serum antibodies against hepatitis B surface antigen were measured by radioimmunoassay (Abbott). Only a single investigator was able to link serologic results with subject identifiers.

The New York City metropolitan area was defined to include Nassau, Suffolk, and Westchester Counties, and areas of New York State, New Jersey; and Connecticut within 25 miles of New York City. The principal type of practice was defined as the area of practice in which at least 50 percent of professional time was spent.

Subjects who reported parenteral drug abuse and men who reported having sexual relations with other men in the years since 1975 were analyzed separately.

RESULTS

Two hundred sixty-six persons who declined to participate in the study anonymously completed refusal forms indicating one or more reasons for nonparticipation. Reasons given for nonparticipation included a lack of time (78 subjects), a need to think about the study more (68), concern about the maintenance of confidentiality (61), not wanting a venipuncture (51), being in a group at high risk for AIDS and preferring not to be tested (15), a belief that the research project was not important (9), and other, including being retired, "knowing" that dentists were at high risk, "knowing" that dentists were at no risk, a belief that the serologic tests are inaccurate, and fear of revocation of their dental license if they were found to be positive for HIV (62).

A total of 1360 subjects participated in the study. Blood specimens were not obtained from 13 subjects, who were excluded from further analysis. Also excluded were 25 subjects with no or uncertain patient contact. Thirteen subjects who reported high-risk behavior (10 homosexual or bisexual men, 2 heterosexual intravenous drug users, and 1 drug user who was homosexual or bisexual) were analyzed separately. The remaining 1309 subjects included 1132 dentists, 131 dental hygienists, and 46 dental assistants.

The sex and the location, type, and duration of dental practice of the subjects are shown in Table 1. The majority of the dentists were men; almost all the dental hygienists and assistants were women. Approximately half the subjects worked in New York City, the New York City metropolitan area, or other cities in which large numbers of cases of AIDS have been reported. The majority of dentists practiced restorative dentistry. Fifty-three (5 percent) were oral surgeons, and an additional 461 (41 percent) spent from 10 to 49 percent of their time performing oral surgery. The mean number of years in practice for all subjects was 18.

The number of subjects who reported treating patients known to have AIDS or to be at an increased risk of AIDS and the estimated numbers of such patients treated are shown in Tables 2 and 3. Fifteen percent of subjects treated patients with AIDS; 72

percent treated patients known to be at increased risk for AIDS. Some subjects treated hundreds of such patients. Because subjects were asked to estimate the numbers of their patients known to belong to each of the high-risk groups, it is likely that the estimated numbers were lower than the actual number of high-risk persons treated.

Use of recommended infection-control precautions[33] by subjects in the study was inconsistent; 31 percent of dentists, 73 percent of hygienists, and 8 percent of dental assistants always used gloves when

Table 2. Dental Professionals Who Treated High-Risk Patients

Patient Group	Dentists (N = 1132)	Hygienists (N = 131)	Assistants (N = 46)	Total (N = 1309)
		no (%)		
Patients with AIDS	176 (16)	15 (11)	8 (17)	199 (15)
Persons at risk for AIDS	822 (73)	98 (75)	29 (63)	949 (72)
Homosexual or bisexual men	738 (65)	94 (72)	28 (61)	860 (66)
Intravenous drug abusers	395 (35)	32 (24)	17 (37)	444 (34)
Persons with hemophilia	280 (25)	24 (18)	6 (13)	310 (24)

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Table 3. Estimated Numbers of High-Risk Patients Treated within Five Years

	Dentists	Hygienists	Assistants	All
	median no. (range) of patients treated			
Patients with AIDS	2 (1-75)	1 (1-6)	1 (1-3)	2 (1-75)
Homosexual or bisexual men	10 (1-700)	14 (1-300)	5 (2-250)	10 (1-700)
Intravenous drug abusers	10 (1-800)	2 (1-150)	3 (1-10)	10 (1-800)
Persons with hemophilia	2 (1-100)	2 (1-25)	3 (1-10)	2 (1-100)

treating patients, whereas 7 percent, none, and 10 percent, respectively, never used gloves. The remainder (62 percent of dentists, 27 percent of hygienists, and 82 percent of assistants) used gloves intermittently, either during specific procedures or when specific patients were perceived as being likely to have or be at an

increased risk for a communicable infection. Likewise, the use of eye protection, masks, and disposable gowns was intermittent; the proportions of all subjects who never used these precautions were 8, 9, and 61 percent respectively. Ninety-eight percent of subjects who used precautions reported that they had increased their use of precautions since 1983, and 87 percent had done so completely or partly because of concerns about AIDS.

Accidental parenteral inoculations with sharp instruments were common, as shown in Table 4. Some subjects estimated that hundreds of such events had occurred, with their number ranging up to 7500 over the preceding five years in one dentist.

The results of serologic tests for hepatitis B in the subjects are shown in Table 5. A majority of subjects in all groups had not received the hepatitis B vaccine, and 21 percent of these unvaccinated subjects had antibodies to hepatitis B surface antigen. Since other markers for hepatitis B infection were not measured, the observed seropositivity rates for hepatitis B are the minimum rates of hepatitis B infection among these subjects.

Several subjects had potential nonoccupational exposures to HIV infection. Forty-nine male subjects (4.9 percent) reported having had sexual relations with prostitutes in the years since 1978; the mean number of contacts was 5 (range 1 to 50). Thirty-nine subjects (3.0 percent) had received one or more blood transfusions since 1978. Eight subjects (0.6 percent) reported having had sexual relations with one or more persons at increased risk of AIDS.

Table 4. Accidental Parenteral Inoculations with Sharp Instruments.

	Dentists (N = 1132)	Hygienists (N = 131)	Assistants (N = 46)	Total (N = 1309)
No. (%) reporting accidental inoculations*	765/816 (94)	93/98 (95)	40/46 (87)	898/960 (94)
Median estimated number (range)				
Within five years	10(1-7500)	10 (1-125)	10 (2-1440)	10 (1-7500)
Within one year	3 (1-600)	2 (1-52)	5 (1-288)	3 (1-600)
Within one month	1 (1-20)	3 (1-15)	3 (1-25)	1 (1-25)

*The denominators indicate the number of subjects in each group responding to the question.

Only 1 of 1309 subjects not at increased risk of AIDS because of high-risk behavior had antibody to HIV confirmed by Western blotting (Table 6). This subject was subsequently interviewed confidentially, and a second serum specimen was obtained and confirmed to be seropositive for HIV. He was a dentist who had been practicing restorative dentistry in Manhattan for 14 years. His only sexual contact for more than 10 years had been his wife. He said he had not had sexual relations with men, had not used illicit drugs, and had not received a transfusion of blood products. He had never treated a patient known to have AIDS but had treated persons known to be at increased risk for AIDS. His use of gloves, masks, and other precautions was intermittent, and before February 1985, when he increased his use of gloves because of concerns about AIDS, his use of precautions had been less frequent. He reported that he frequently practiced without gloves, even though he often had obvious breaks in his skin. He estimated that he had received two accidental parenteral inoculations within the previous year and 10 within five years. He had not received hepatitis B vaccine and he had a negative test for antibody to hepatitis B surface antigen. The dentist's wife agreed to a separate confidential interview. She said she had not had sexual contact with anyone other than her husband for more than 10 years and that she had never used illicit drugs or received transfusions of blood or blood products. She refused to be tested for HIV antibody.

DISCUSSION

HIV and hepatitis B infections occur with increased frequency in sexually active homosexual men,[1-3, 20-22] intravenous drug abusers,[8, 22,23] recipients of blood products,[5-7, 24-27] infants born to infected mothers,[9-11, 29] and heterosexual sexual contacts of infected persons.[4,28] HIV and hepatitis B are both found in blood,[12,13, 25, 26] semen,[34, 35] and saliva.[35-39] Therefore, the routes by which HIV may be transmitted appear to be similar to those known to transmit hepatitis B.

The similar epidemiologic features of HIV and hepatitis B and the recognized increased occupational risk of hepatitis B

infection among health care workers[40] have generated considerable concern among these professionals. Recent reports of HIV infections in nine health care workers that were apparently acquired occupationally as a result of accidental parenteral needle-stick injuries,[41-44] of extensive contact with blood and body fluids in the absence of recommended barrier precautions,[45, 46] and of accidental spills of blood on mucous membranes or abnormal skin,[47] have heightened this concern. However, it is important to recognize that although anecdotal reports may demonstrate that an occupational risk exists, they cannot define a rate of risk; such a definition would re-

[Add. 110]

[Table 5 omitted]

quire studies of large numbers of health care workers exposed to persons with HIV infection or body fluids of such persons. Several studies of health care workers have shown the risk of nosocomial HIV infection to be low. In five studies of occupational risk for HIV infection among health care workers that included 1673 subjects, most of whom were hospital workers caring for patients with AIDS, 770 of the subjects had had parenteral needle-stick injuries or contamination of mucous membranes or open wounds by body fluids from patients with AIDS. Only 8 of 1673 (0.18 percent) became infected with HIV.[48-52]

Dental professionals represent an important group to study for occupational risk of HIV infection. Dentists are among the health care professionals at highest risk of hepatitis B infection, with the risk for oral surgeons being particularly high.[30,31,53-56] Dental hygienists and dental assistants are also at increased risk.[56] Dental professionals are likely to be exposed repeatedly to persons infected with HIV, often for months or years before those persons become ill or know that they have an HIV infection. In addition, dental professionals are at risk not only for accidental parenternal inoculations but also for splashes and aerosolization of blood and

saliva. Therefore, it is important to study this group of health care workers not only to define their risk of HIV infection but also because they may be considered a "sentinel" population that is likely to indicate the maximal anticipated rate of occupational risk for health care workers in general.

Although a substantial number of persons chose not to participate in this study, the subjects who were enrolled were similar to those in previous studies of dental professionals in that only a minority had been vaccinated against hepatitis B,[57] there was a high rate of previous hepatitis B infection among those not vaccinated,[30,31,53,56] their use of recommended infection-control precautions was intermittent,[56-59] they had had frequent accidental parenteral inoculations with contaminated instruments,[56-59] and they had recently increased their use of precautions in response to concerns about AIDS.[59]

Table 6. Antibodies to HIV among Dental Professionals

Group	No. Screened	No. positive (%)
No. high-risk behavior	1309	1 (0.08)
Dentists	1132	1 (0.09)
Dental hygienists	131	0
Dental assistants	46	0
High-risk behavior*	13	4 (31)

*Male homosexual or bisexual activity, intravenous drug abuse, or both.

This study demonstrates that despite infrequent compliance with recommended infection-control precautions, frequent occupational exposure to persons at increased risk for HIV infection, and frequent accidental parenteral inoculations with sharp instruments, dental professionals currently are at low occupational risk for HIV infection. Only 1 subject among 1309 without behavioral risk factors for AIDS had antibodies to HIV. The absence of other risks, as determined by the history obtained from this dentist and his wife, suggests that occupational exposure was the likely mode of acquisition of his HIV infection. If this assumption is accurate, then the observed risk among all subjects

was 1 in 1309 (95 percent confidence interval, 0 to 0.004), the risk among all subjects practicing in locations with a large number of cases of AIDS was 1 in 673 (95 percent confidence interval, 0 to 0.008), and that among dentists practicing in these locations was 1 in 523 (95 percent confidence interval, 0 to 0.011). In an additional 529 dental workers studied in three separate investigations, no HIV infections were observed.[59-61]

Patients who may present a risk of transmission of HIV to dental professionals cannot be detected reliably.[33] Infected persons may not feel or appear ill. They may not consider themselves at high risk of infection or may choose not to inform the dental professional about high-risk behavior. Available serologic tests take time, often require confirmatory testing, and may yield false negative results. In most locations, regulations require voluntary informed consent for HIV testing, and withholding care from patients who refuse to be tested could be considered coercive. Therefore, routine practices to prevent occupational infection should be formulated with the idea that all patients have the potential to transmit infection. This will be particularly important as the prevalence of HIV infection among dental patients increases over time, and potential occupational exposures become more frequent. Strict adherence to recommended infection-control guidelines for dental professionals[33] should help to minimize the risk of occupationally acquired HIV infection.

[Acknowledgments omitted]

[Add. 111]

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[Add. 112]

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Transmission of Human Immunodeficiency Virus in a Dental Practice

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■ *Objective*: To determine if patients of a dentist with the acquired immunodeficiency syndrome (AIDS) became infected with human immunodeficiency virus (HIV) during their dental care, and if so, to identify possible mechanisms of transmission.

■ *Design:* Retrospective epidemiologic follow-up of the dentist, his office practice, and his former patients.

■ *Setting:* The practice of a dentist with AIDS in Florida.

■ *Participants:* A dentist with AIDS, his health care providers and employees, and former patients of the dentist, including eight HIV-infected patients.

■ *Measurements:* Identification of risks for HIV transmission (if present), degree of genetic relatedness of the viruses, and identification of infection control and other office practices.

■ *Results:* Five of the eight HIV-infected patients had no confirmed exposure to HIV other than the dental practice and were infected with HIV strains that were closely related to those of the dentist. Each of the five had invasive dental procedures, done by the dentist after he was diagnosed with AIDS. Four of these five patients shared visit days ($P > 0.2$). Breaches in infection control and other dental office practices to explain these transmissions could not be identified.

■ *Conclusion:* Although the specific incident that resulted in HIV transmission to these patients remains uncertain, the epidemiologic evidence supports direct dentist-to-patient transmission rather than a patient-to-patient route.

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In July 1990, the Centers for Disease Control (CDC) published the first of a series of reports describing human immunodeficiency virus (HIV) transmission from a health care worker to a patient (1-3). No case of such transmission had been previously reported, although transmission of another blood-borne pathogen, hepatitis B virus (HBV), from infected health care workers to patients has been well documented, and the theoretic possibility that HIV transmission could also occur had been previously acknowledged (4-6). These reports intensified the ongoing debate concerning whether HIV and HBV-infected health care workers should do invasive medical and dental procedures. Resolution of this question has been hampered by the lack of accurate quantitation of the risk for transmission and also by misinformation and misconceptions regarding the likely mechanism of HIV transmission in the dental practice.

We review updated epidemiologic findings which led to the conclusion that five patients of a dentist with the acquired immunodeficiency syndrome (AIDS) were infected with HIV during their dental care. We also discuss the possible mechanisms of disease transmission.

Methods

The investigation began when a young woman without identified risk factors for HIV infection (Patient A) was reported to have AIDS. During a routine health department follow-up interview, patient A provided the name of her dentist who, 2 years previously, had extracted her maxillary third molars. She was aware that the dentist had become ill and suspected that he might have AIDS. The dentist was known to the Florida Department of Health and Rehabilitative Services investigator as a previously reported AIDS case.

After publication of a report describing the possible HIV transmission to Patient A, the dentist publicly requested that his former patients seek HIV testing. The dentist was no longer in practice, and he could not provide a complete list of his former patients. However, a partial list was compiled from names provided by insurance companies. Medicaid records, billing and other records from the practice, and from other dental offices in the area. Patients who had not been tested for HIV at the local county public health unit were notified by the Florida Department of Health and Rehabilitative Services. By the end of 1991, 2 of the approximately 1100 former patients tested were found to be HIV-seropositive (Patients B and C).

A fourth patient (Patient D) was identified by confidentially matching the names of the former patients to the state's reported AIDS cases. Three former patients (Patients E, G, and H) notified the CDC that they were infected with HIV. Another patient (Patient F) was identified during the epidemiologic investigation of Patient E.

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Table 1. Characteristics of the Dentist and the HIV-infected Patients Identified in the Dental Practice*

Patient	Identified Risk Factor for HIV	CD4 +/ μL†	MHA=TP FTS-ABS†	HBsAg†	Anti-HB_c†
A	No	< 50	Negative	Negative	Negative
B	No	222	Negative	Not done	Negative
C	Unconfirmed	< 50	Negative	Not done	Negative
D	Yes	Not done	Negative	Negative	Positive
E	No	567	Negative	Negative	Negative
F	Yes	253	Negative	Negative	Negative
G	No	400	Negative	Negative	Negative
H	Yes	640	Negative	Positive	Positive
Dentist	Yes	190‡	Negative	Negative	Positive

*CD4+= CD4 lymphocytes: FTA-ABS = fluorescent *Treponema pallidum* antibody absorption: HBsAg = hepatitis B surface antigen: anti-HBc

= antibody to hepatitis B core antigen: MHA-TP = microhemaggiutination assay for antibodies to *Treponema pullidum*.
┼ At initiation of epidemiologic investigation.
‡ At time of AIDS diagnosis.

Epidemiologic Investigations of the Patients and Dental Practice

The patient investigations included interviews with the patients and with their families and friends; discussions with their health care providers about possible exposures to HIV; review of medical, dental, and related records; and serologic testing of the patients' sex partners.

In March 1990, the dentist permitted a single interview with the CDC. During this interview, the dentist was informed that one of his patients had been diagnosed with AIDS and that this patient had no identified risk factors for HIV infection. With the dentist's consent, a blood sample was drawn for DNA sequence analysis to determine the degree of relatedness of his virus to that of Patient A. Information about the dentist's medical history was obtained from interviews with his health care providers and family and from a review of his medical and dental records.

Employees of the dental practice were tested for HIV infection and were interviewed on multiple occasions regarding infection control and other work practices in the dental office. The dental office was inspected in October 1990, approximately 14 months after it had been sold to another dentist.

Laboratory Investigation

Human immunodeficiency virus strains from persons with an epidemiologic transmission link have been shown to exhibit a closer genetic relatedness than do strains from persons without such a link (7-9). To determine the relatedness of the viruses, blood specimens were obtained from the dentist and from the eight infected patients. Samples from 35 HIV-infected patients receiving care at clinics within the same geographic area as the dental practice served as controls. Computer-based methods including genetic distance measurements and amino acid signature pattern

analysis were used to compare the DNA sequences of an approximately 240-nucleotide portion of the C2-V3 domain of the dentist's virus to that of the other viral strains (50). The C2-V3 domain of the envelope gene was chosen because this domain contains nucleotide sequences with sufficient variability to distinguish strains and because this region has been used in previous studies of epidemiologically linked infections (7-9).

Results

Characteristics of the eight infected patients identified in the dental practice are summarized in Table 1. An illness possibly compatible with acute retroviral syndrome was found only for Patients A and F. No patient received blood or blood products.

Patient A denied injecting drugs, and her two reported boyfriends before her AIDS diagnosis tested seronegative for HIV in 1990. Her six dental visits, which occurred between November 1987 and June 1989, included one examination, two prophylaxes, two cosmetic bondings, and one visit for extraction of her maxillary third molars (Table 2). The patient recalled that the dentist wore gloves and a mask during the procedures. Dental x-rays taken during the spring of 1990 showed no additional extractions.

Medical records indicated that 4 weeks after the dental extraction, she was evaluated for a sore throat. She was afebrile, and her ulcerated tonsils and anterior cervical lymph nodes were moderately enlarged. The diagnosis was pharyngitis and aphthous ulcers; no indication exists that an acute retroviral syndrome was considered. She was diagnosed with oral candidisis in May 1989 (17 months after the extraction) and with AIDS in December 1989 (24 months after the extraction), when she developed *Pneumocystis carinii* pneumonia. At that time, she was first tested for HIV antibody and was found to be seropositive.

Patient B reported that her only sex partner since 1978 was her husband, who tested seronegative for HIV. She denied injecting drugs.

Her 21 dental visits, between December 1987 and July 1989, included four extractions (*see* Table 2). Other procedures included three prophylaxes, three periodontal scalings, one examination, two restorative fillings, and ten fixed and removable prosthodontics. She recalled that the dentist wore gloves and a mask. She also recalled cutting her tongue on a tooth that was "sharply filed" during preparation for a bridge; however, she was unaware of any injury that the dentist may have sustained at that time.

Patient C denied having sex with men and injecting drugs. He reported multiple heterosexual partners but could name only 14 former female sex partners since 1978, and he was unaware that any were infected with HIV. Nine of these former partners were located and were found to be HIV seronegative. The possibility that Patient C had engaged in high-risk behaviors was raised during interviews and record reviews; however, behavioral exposures to HIV could not be documented.

He made 14 visits to the dental office between December 1984 and May 1989; the procedures included two extractions, two examinations, six restorative fillings, four prophylaxes, one periodontal scaling and one

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Table 2. Dental Appointment Dates for Patients A, B, C, E, and G*

Year	January	February	March	April	May	June	July	August	September	October	November	December
1987											(A(16)	A&B(17)+ B(21)
1988					B(12)	S(18,23,30)	B&E11+ E(5,28) G(18,25)	B(1,4,8,15, 16,18)	B(1,8,23,26) E(2,9,19)	B(20) E(8,7,11)	B(8)	A(23,29,30) C(6) E(8)
1989	B&C(20)+ C(10,16)		B(10)	C(19)	A(29)	B(21,27)	Practice closed					

*Letters indicate patients: numbers in parentheses indicate day of the month.
+Shared-visit days

cosmetic bonding (*see* Table 2). Nine of these visits, including both of the extractions, occurred in 1984 and 1985, before the dentist began to wear gloves routinely (Figure 1). The patient did

recall that the dentist wore gloves and a mask during his visits in 1988 and 1989.

Patient D, a man who had behavioral risks for HIV infection, was diagnosed with AIDS in 1989. He had 18 visits to the dental office between June 1985 and May 1989. The procedures included five examinations, five extractions, six prophylaxes, two periodontal scalings, four removable prosthodontics, and two restorative fillings.

Patient E denied injecting drugs. Of her ten known sex partners since 1978, two had died from non-HIV-related causes and were not known to be at risk for HIV infection. Each of the remaining eight was tested for HIV antibody; one was seropositive. This man (Patient F) had other risk factors for HIV infection and was also a patient of the dentist. Patient E reported infrequent sexual contact with Patient F, the last contact occurring in the fall of 1988. Because of her concern about Patient F's high-risk behavior, she was first tested for HIV antibody in October 1988. Patient E was seropositive, whereas Patient F was seronegative.

Billing records and receipts indicated that Patient E made 14 visits to the dentist between June 1988 and December 1988 (*see* Table 2). The procedures included four examinations and radiographs, two prophylaxes, two root canal therapies, one restorative filling, six fixed prosthodontics, and one fluoride treatment. She reported that the dentist wore gloves and a mask during her visits.

Patient F tested negative for HIV antibody in October and December 1988, but he tested positive during the epidemiologic follow-up investigation in December 1990.

In September 1989, his medical records indicate a 1-week history of sore throat, loose stools, and headaches; other symptoms included decreased appetite, fatigue, myalgia, and an earache. He had a temperature of 38.1°C and tender anterior cervical adenopathy; his leukocyte count was 3300/μL. A diagnosis of tonsillitis was made, and a throat culture yielded "normal respiratory flora." No HIV-antibody test was done at that time, nor

does any indication exist that an acute retroviral syndrome was considered. This illness occurred approximately 1 year after Patient F's last reported dental appointment (August 1988) and his last sexual contact with Patient E (August 1988) and 9 months after his last negative HIV antibody test (December 1988).

Review of Patient F's medical and other records established a history of behavioral risk factors for HIV infection. Only one dental visit was documented by billing records, although he reported five or six visits during July and August 1988 for examination, radiographs, prophylaxis, extraction, restorative fillings, and root canal therapy.

Patient G denied, since 1978, sex with men and injecting drugs. He first tested positive for HIV during screening for plasma donation in November 1990. He had been HIV seronegative when he donated blood in 1986, and his two reported female sex partners since 1986 were HIV seronegative.

Patient G's dental records could not be located, but records from an alcohol rehabilitation facility documented two visits in July 1988 for root canal therapy and a restorative filling (*see* Table 2). He could not recall whether the dentist wore gloves or a mask.

Patient H has acknowledged behavioral risks for HIV infection. He made three visits to the dentist between September 1985 and July 1986 for prophylaxes, examination and gingivectomy.

With the exception of patients E and F, none of these patients named each other as a sex partner, and all denied sexual contact with the dentist. For all of the patients, local anesthesia was the only anesthetic administered, and none could recall an injury to the dentist that would have exposed them to his blood (for example, a needlestick or a cut with a sharp instrument).

Laboratory Findings on Patients

The nucleotide sequences of the C2-V3 domain of the viruses from Patients A, B, C, E, and G were closely related to

those of the dentist's virus. The average nucleotide variation ranged from 3.4% to 4.9% (50), a range similar to those previously observed for HIV strains from persons whose infections were known to be epidemiologically linked (7-9). In contrast, the viruses from Patients D, F, and H, each of whom had high-risk behaviors, were not closely related to the dentist's virus, and differed by 13.6% and 10.7% for Patients D and F, respectively (50). This degree of genetic difference has been found among strains of HIV that were epidemiologically unrelated. Preliminary analysis indicates that the virus from Patient H is also not closely related to that of the dentist. No instance of comparable viral

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sequence relatedness was identified among the viruses from the 35 local controls (50). The HIV strains from patients A, B, C, E, and G were significantly closer in their DNA sequences to the dentist's virus than control patients ($P = 6 \times 10^{-6}$, Wilcoxon rank-sum test) (50).

An unusual pattern or signature of eight noncontiguous amino acids encoded by V3 nucleotides was found in the dentist's virus. The viruses from Patients A, B, C, E, and G, had at least 7 of the 8 signature amino acids. This degree of agreement was not found in the viruses from the dental patients with behavioral risks for HIV infection, the local controls, or a reference set of sequences from 32 HIV-1 strains ($P = 8 \times 10^{-6}$, Wilcoxon rank-sum test) (50). This finding, together with the genetic distance analysis, suggests that the virus from the dentist and those from Patients A, B, C, E, and G formed a cluster of genetically similar strains that were distinct from the viruses of the patients with identified risks for HIV infection and from the viruses of 35 local control patients.

Interview with the Dentist

The dentist recalled having sustained needlestick injuries from anesthetic needles before he was diagnosed with HIV

infection, but did not recall sustaining any percutaneous injuries after learning he was HIV positive.

The dentist reported nonoccupational exposures to HIV, and he believed that he had been infected through these exposures, although the exact time of infection remains unknown. He was unaware that any of his patients were infected with HIV, although he suspected that as many as ten patients may have been at risk for HIV infection. He denied sexual contact with any of his patients.

Medical History of the Dentist

Review of the dentist's medical records showed that he was diagnosed with symptomatic HIV infection in late 1986 and with AIDS in September 1987, as indicated by a biopsy result diagnostic of Kaposi sarcoma of the palate and a CD4 + lymphocyte count of 190/μL. Zidovudine therapy was begin in early November 1987 but was discontinued from 16 December through 23 December 1987 because of neutropenia (*see* Figure 1). It was then continued at different doses throughout the time he remained in clinical practice.

In May 1988 examination of the palate showed purplish discoloration of the mucosa of the entire hard palate without ulceration or exophytic growth. The remainder of his physical examination was unremarkable. From 6 June through 20 June 1988, he received a total of 2750 rads of radiation therapy to his palate. This treatment resulted in mucositis at the end of therapy. By 6 July 1988, the palatal lesions had regressed, and the mucositis reaction had subsided. According to his medical records and interviews with his health care providers, he remained relatively asymptomatic except for fatigue until March 1989, when several lesions indicative of Kaposi sarcoma were noted on his abdomen and thorax. In June 1989, he was hospitalized with a presumptive diagnosis of *P. carinii* pneumonia. According to medical records, in the summer of 1989 his cutaneous Kaposi sarcoma lesions were treated with liquid nitrogen, and he reported that he used an electrocautery unit kept at his home to self-cauterize some of the lesions. In the summer of 1989, he stopped practicing dentistry. During the time he was in practice, his medical records did not

indicate thrombocytopenia or any other bleeding disorder, hand dermatitis, injury, or dementia. No indication of peripheral neuropathy was present, although no record of a detailed neurologic examination or testing for neurologic dysfunction was found in the medical records.

In October 1989, the Kaposi sarcoma progressed to involve his lungs, and he was treated with alpha-interferon and chemotherapy. He died in September 1990.

The Dental Practice

The dentist had a general practice at one location from 1981 to 1989. He was in solo practice except from September 1985 through February 1986, when he had an associate. In the summer of 1989, the office was sold to another dentist. The patient records were dispersed and many office records, such as appointment books, were

[Figure 1 omitted]

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discarded. The office was remodeled, and most of the dental equipment was replaced. The office staff stated that there had been no electrocautery unit in his office practice.

All 16 persons identified as having been employed in the dental practice, including receptionists, hygienists, and assistants, were located and interviewed. All tested seronegative for HIV antibody. Staff members were not aware of any patients who were known to be infected with HIV or of any who had been sex partners of the dentist.

Staff members reported that since 1987, the dentist had received one prophylactic treatment from the hygienist and on one or two occasions had had an unspecified liquid medication placed on a palatal lesion using a cotton-tipped applicator that was then

discarded. To the knowledge of the staff members, the dentist received no other treatment and did not perform any treatment on himself in his office.

Infection Control Practices

Barrier precautions were fully introduced into the practice by early 1987; all staff with patient contact wore latex gloves and surgical masks for patient-care activities when appropriate. On most occasions, gloves were changed and hands were washed between patient contacts. Occasionally, however, gloves were washed rather than changed. Masks reportedly were changed infrequently. No staff member reported noticing any dermatitis or other lesions visible on the dentist or witnessing the dentist sustain a percutaneous injury during patient treatment.

By 1987, all surgical instruments used in the practice were autoclaved; other heat-tolerant instruments were autoclaved when time and instrument supply allowed or were immersed in a 2% glutaraldehyde solution for varying periods, usually of at least 20 minutes duration. Dental equipment such as prophylaxis angles and air and water syringe tips were either wiped with alcohol or immersed in 2% glutaraldehyde after each patient encounter. Some disposable items, such as saliva ejectors, high-speed evacuation tubes, and prophylaxis cups, were occasionally reused after being immersed in 2% glutaraldehyde, usually for at least 20 minutes. Handpieces were wiped with alcohol after each patient encounter; water lines were not flushed between patients.

The dental practice had no written infection control protocol or consistent pattern for operatory cleanup and instrument reprocessing. No office protocol existed for reporting or recording injuries such as needlesticks or other percutaneous injuries involving sharp instruments or devices.

Only local anesthetic was used in this practice, and anesthetic needles were either recapped by the dentist using a two-handed technique or remained uncapped and were recapped by the assistant on completion of the dental procedure. By 1983, only single-use disposable anesthetic carpules and disposable needles

were used. Staff members reported no instance in which a needle or carpule was reused on different patients; however, the same needle was routinely used multiple times on the same patient when multiple injections of local anesthetic were required. The dentist did not document his use of local anesthetic in patient records.

Days of Shared Visits

On four occasions, two of the five infected patients whose viruses were related to that of the dentist were seen in the dental office on the same day (*see* Table 2, Figure 1). Those with shared visit days included Patients A, B, C, and E: Patient G did not share any visit days with the other four patients.

The five patients made a total of 48 visits to the dental office from November 1987 until the close of practice in July 1989. To examine the likelihood that the five patients shared visit days, conditional probabilities were calculated based on the number of visits made by each patient between November 1987 and July 1989, the interval during which at least two of these five HIV-infected persons were patients of the dentist. The probabilities were calculated assuming that visits occurred during days on which the dentist's office was open, without allowing multiple visits for the same patient on the same day. These shared-visit days probably occurred by chance alone ($P > 0.2$). Because appointment books no longer existed, neither the order nor the time of day could be determined for any of the shared-visit days.

Patients A and B were seen on 17 December 1987 (*see* Table 2). Patient A had two extractions while receiving local anesthetic. Patient B was examined for a toothache, and a radiograph was taken: no invasive procedures were done, although she had an extraction 4 days later. No indication exists that a handpiece was used on either of these patients on this day.

On 11 July 1988, Patient B had periodontal scaling and an extraction. Patient E had a crown placed on a maxillary bicuspid tooth. Patient B received local anesthetic, but we could not determine whether an anesthetic was used for placement of Patient E's crown because the tooth had previously had a completed root

canal. It is unlikely that instruments used for the periodontal scaling and extraction would also be used to place a crown.

On 29 August 1988, Patient B had a bridge placed, and Patient E had plastic restorations placed on two maxillary anterior teeth. Although it is probable that local anesthetic was used on both patients on this day, the other instrumentation would differ. We could not determine whether the dental handpiece was used during the placement of the bridge; however, it would have been used for the restorations on patient E.

On 20 January 1989, Patients B and C had prophylaxes. Patient B's prophylaxis was done by the dental hygienist; however, we could not determine whether Patient C's prophylaxis was performed by the dentist or by the hygienist. It is unlikely that either patient had local anesthetic administered on that day.

Discussion

The findings of this investigation led us to conclude that five patients of a dentist with AIDS became infected with HIV during their dental care. We support

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this conclusion because the five patients had no other confirmed exposures to HIV; all had invasive procedures done by the dentist; and DNA sequence analyses of the HIV strains from these five patients indicate a high degree of similarity among the strains and that which infected the dentist. These HIV strains were distinct from those infecting the three patients in the practice who had identified risk factors for HIV infection and from 35 HIV-infected persons residing in the same geographic area.

Our investigation is the first to report the transmission of HIV to patients during receipt of health care, although HIV transmission from infected patients to health care workers is well documented (10-12). In addition, transmission of HBV, another

blood-borne virus, from infected health care workers to patients has been reported repeatedly since the early 1970s. More than 330 cases of HBV transmission from health care workers to patients occurring during invasive procedures have been reported (13-32) (Shapiro CN, CDC, unpublished data). Virtually all cases have involved clusters associated with care by 33 HBV-infected health care workers, including 9 dentists and 20 surgeons. Although percutaneous transfer of blood was the most likely mechanism of HBV transmission from health care worker to patient, the specific incident which resulted in transmission was not identified in any of the 33 episodes investigated.

In none of these studies involving an HBV-infected surgeon or dentist was patient-to-patient transmission of HBV believed to have been a mechanism of transmission. However, in other healthcare settings, patient-to-patient transmission of HBV has been attributed to contaminated common-source vehicles, including jet guns, multidose vials of heparin and local anesthetic, and fingerstick devices (33-36).

Although the precise circumstances or mechanism of HIV transmission in this Florida dental office remains unclear, the preponderance of data support direct dentist-to-patient transmission rather than a patient-to-patient route. The dentist continued to do many invasive procedures after he was diagnosed with AIDS. This period includes the interval during which his zidovudine was discontinued and during which a course of radiation therapy was administered. All five patients had invasive procedures done after the dentist was diagnosed with AIDS and had evidence of severe immunosuppression. Low CD4 + lymphocyte counts are associated with higher viral titers in the blood (37) and therefore could be associated with an increased likelihood of transmission if an injury to the dentist occurred. In addition, interviews with the dentist's health care providers and his office staff indicated that, after he was diagnosed with AIDS, he frequently experienced fatigue, a factor which may have increased the likelihood of injury.

There were multiple opportunities for the dentist to have injured himself during invasive procedures done on these patients, especially during periods of fatigue and ill health. In a survey of

self-reported sharps injuries in 89 dentists, 32% reported 2 or more per month, and 3% indicated more than 15 such injuries per month (38). Another survey of 1132 dentists found a median of one sharps injury per month (39). Further, although undocumented, the possibility that the dentist had a peripheral neuropathy cannot be ruled out: peripheral nervous system dysfunction has been noted in 9% to 35% of patients with AIDS (40-43).

All five patients received multiple injections of local anesthetic, and a sharps injury during anesthetic administration could have resulted in contamination of the syringe apparatus with the dentist's blood, after which additional anesthetic may have been injected into the same patient. A sharps injury could also result in direct contact of the dentist's blood with the patient's inflamed or nonintact oral tissue during the invasive procedures. Although the dentist began to routinely wear gloves in 1987, gloves do not prevent most injuries caused by sharp instruments.

Human immunodeficiency virus does not remain viable for extended periods outside the body, and it is susceptible to commonly used germicides, including those used in the practice (4). Interviews of staff members indicate that, before use on patients, all instruments were cleaned and reprocessed by methods known to inactivate HIV, although these methods may not comply with recommendations designed to kill more resistant organisms.

If contaminated instruments or equipment are assumed to be the principal factor in these transmissions, one would expect to see a clustering of patient appointments. Although some shared-visit days were documented, these probably occurred by chance alone. Additionally, the procedures done on these shared visit days probably did not involve the use of the same instruments on more than one of the infected patients. Questions have been raised about the possible role of the dental handpiece in these transmissions. It is unlikely that a high-speed dental handpiece was used on both patients on any of the shared-visit days, and, to date, no studies have confirmed the transmission of blood-borne viruses such as HIV or HBV through the dental handpiece or other dental equipment.

The dentist denied having sex with any of his patients. One HIV-infected person subsequently contacted the CDC and reported having had sex with the dentist in the early 1980s and occasionally receiving dental care from this dentist before moving from the area in 1985. However, no patient records exist to confirm these data. No other persons who were both sex partners and patients of the dentist have been identified.

The possibility that these infections were intentionally transmitted has been hypothesized. Interviews with family, staff, health care providers, patients, and others who knew the dentist have not provided any support for this hypothesis. The dentist initially cooperated with our investigation and provided a blood specimen for genetic sequencing to be compared with patients' specimens. Additionally, most of the procedures done by the dentist were routinely observed by staff, all patients were awake during the procedures, and no unusual behavior was noted or suspected by either patients or staff members.

Some have hypothesized that the dentist might have had an exceptionally high titer of virus or an easily transmittable strain of HIV. The titer of HIV in the dentist's blood obtained in March 1990 is currently be-

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ing determined, although it is unclear how accurately this result would reflect the viral titer in his blood between 1987 and 1989. Strain-specific virulence factors for HIV transmission have not been identified; further, no evidence was found to suggest HIV transmission from these patients to their sex partners.

The five patients identified in our investigation remain the only reported cases of HIV transmission from an infected health care worker to patients during invasive procedures. Results from six other studies of patients exposed to HIV-infected surgeons or dentists have not shown transmission (44-49). Of the 1246 patients of these health care workers who are known to have been tested,

one was positive for HIV infection; however, this patient had behavioral risks for HIV infection and was probably infected before his surgical procedure (48).

The risk for transmission of blood-borne infections, including HBV and HIV, from an infected health care worker to a susceptible patient during an invasive dental or surgical procedure may depend on several factors, including the type of procedure, the infection control precautions, and the individual technique of the health care worker. The relative importance of these and perhaps other factors in predicting the risk for such transmission is poorly understood and requires further study.

Currently available information indicates that when health care workers adhere to recommended infection control procedures, the risk for transmitting HBV from an infected health care worker to patients is small, and the risk for transmitting HIV is probably even smaller. These small risks should not deter patients from seeking necessary surgical or dental care.

[acknowledgments, current author addresses and references omitted]

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Vol. 42 / No. 17 MMWR 329

Current Trends

Update: Investigations of Persons Treated by HIV-Infected Health-Care Workers -- United States

Previous reports have described transmission of human immunodeficiency virus (HIV) to five persons (patients A, B, C, E, and G) during receipt of care from an HIV-infected dentist in Florida (1-3) and other investigations of patients who were treated by HIV-infected health-care workers (HCWs) (4). This report updates these investigations and presents evidence that a sixth patient (patient I) became infected with HIV while receiving care at the Florida dental practice, the only practice in which HIV transmission from an infected HCW to patients has been documented.

Investigation of Patients of HIV-Infected HCWs (Excludes Florida Dental Practice)

As of March 31, 1993, HIV tests were completed for 19,036 persons treated by 57 HIV-infected HCWs. These results include findings in published reports (4-7) and unpublished investigations reported to CDC.

No seropositive persons were reported among 11,529 patients tested from the practices of 46 HCWs, including 23 dentists and dental students, 12 physicians and medical students, seven surgeons and obstetricians, and four others. For the remaining 11 HCWs (six dentists and five surgeons and obstetricians), 7507 patients were tested, and 92 seropositive patients were identified. Follow-up investigations have been completed for 86 (94%) of these 92 patients; eight patients were documented to be infected before receiving care from the HIV-infected HCW; 54 had established risk factors for HIV; 19 may have had other opportunities for exposure to HIV (i.e., exchange of sex for drugs or money and/or multiple sex partners); and five had no risks identified. Investigations are in progress for six patients of two HCWs.

Genetic sequencing was performed on HIV strains from 29 of the 92 seropositive patients from the practices of three HCWs. Eleven of these 29 had established risks, 15 had other opportunities for exposure to HIV, and three had no identified risk. Sequencing was not performed on the isolates for the remaining two of the five patients with no identified risk because one patient died before a blood sample could be collected, and the other refused to provide a sample. The degree of genetic similarity of viruses from the patients and the infected HCWs was in the range previously reported for persons with epidemiologically unrelated infections (5,6; CDC, unpublished data). Thus, follow-up to date has not demonstrated transmission from an HCW as a source of HIV infection for any of the patients tested.

Epidemiologic and Laboratory Investigation of Patient I

Patient I, a teenaged female, was HIV seropositive when tested as an applicant for military service in late 1992. She had not previously been tested for HIV infection, although she was notified in December 1990 by the Florida Department of Health and

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Rehabilitative Services (HRS) that, as a former patient of the dentist, she should consider such testing.

Multiple interviews with the patient and her family and review of her medical records did not identify another mode of exposure to HIV. She denied previous injecting-drug use, receipt of blood or blood products, a history of sexually transmitted diseases, or sex with persons infected with HIV or at increased risk for HIV infection. She did not recall, nor did review of her medical records reveal, an illness compatible with an acute retroviral illness. Five of her six lifetime sex partners were tested and were negative for HIV antibody. The sixth sex partner, with whom the patient

reported a single sexual contact using a condom, has not been located. The patient's CD4+T-lymphocyte count at the time of HIV diagnosis was 429 cells/μL. Serologic tests for syphilis and hepatitis B were negative.

Interviews with the patient and her parents indicated she was a patient in the dental office during the summers of 1987, 1988, and 1989 for examinations, radiographs, prophylaxes, and restorative fillings under local anesthesia. Her dental records from the practice cannot be located; therefore, whether she shared a visit date with any of the other five infected patients is unknown. An insurance record documented a visit in August 1988 for an examination, radiographs, and prophylaxis. Bitewing radiographs taken in 1990 by another dentist indicate that single surface restorative fillings had been placed in three permanent molars. Before 1987, the patient had received no other dental care since the eruption of her permanent teeth. She did not recall any injury to the dentist or other unusual events during the dental procedures or whether the dentist or the hygienist performed the prophylaxes.

Peripheral blood mononuclear cells were obtained from patient I, and proviral DNA was extracted, amplified, cloned, and sequenced (2) to determine the relatedness of the HIV strain from patient I to those of the dentist and patients A, B, C, E, and G. A direct sequence of amplified DNA and nine cloned sequences each included approximately 325 nucleotides of the C2-V3 region of the *env* gene. In addition, six shorter clone sequences were produced. Sequence analyses were performed at Los Alamos National Laboratory and at CDC.

The genetic divergence (i.e., intrapatient nucleotide sequence distances) among the nine complete C2-V3 clones from patient I averaged 3.2% (range: 0.3%-5.0%). The viruses of the dentist and patient I were closely related, with an average genetic divergence of 4.3% (range: 2.8%-7.8%), and the viruses of patient I and patients A, B, C, E, and G also were closely related, with an average divergence of 4.9% (range: 2.1%-8.1%). In comparison, the sequences of patient I were distinct from those of the 34 local controls (2), with an average genetic divergence of 12.0% (range: 6.8%-17.9%). Based on direct sequences of 186 nucleotides from

the viruses infecting patients A, B, C, E, and G were significantly closer in their DNA sequences to patient I's virus than the control patients ($p<0.0001$, Wilcoxon rank sum test).

In addition to the genetic divergence analysis, signature pattern analysis of both amino acids and nucleotides* (2,8) and phylogenetic tree analysis (2) each independently showed the similarity of patient I's sequences and those of the dentist and patients A, B, C, E, and G. Analyses of the six shorter clone sequences and the direct amplification product from patient I were consistent with this finding.

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HIV Infection -- Continued

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Editorial Note: The results of the epidemiologic and laboratory investigation of patient I indicate that she became infected with HIV while receiving care from an HIV-infected dentist. She had had no other confirmed exposures to HIV. In addition, DNA sequences analysis showed her HIV strain had a high degree of similarity to that of the dentist and the five other infected patients. The precise event(s) resulting in HIV transmission in this practice remain(s) unknown(1). Unlike the other five infected patients, patient I had neither dental extractions nor root canal therapy. Opportunities for injuries to the dentist were limited. However, exposure of patient I to the dentist's blood cannot be ruled out (e.g., related to use of the anesthetic syringe).

Approximately 1100 patients of the dentist are known to have been tested for HIV. In late 1990, HRS used available records

to compile a partial list of the patients in the dental practice and notified those patients who had not been tested. Former patients who have not yet been tested should contact their local health department or private physician to discuss HIV testing.

Among the 58 investigated practices described in this report, the dental practice in Florida remains the only documented instance of HIV transmission from an HCW to patients. The risk for transmission of a bloodborne pathogen from an HCW to a patient is associated with the circulating titer of the pathogen in blood, the procedures performed, techniques and infection-control precautions used, and the medical condition of the HCW (9).

The results presented in this report are consistent with previous assessments that the risk for HIV transmission from an infected HCW to patients during invasive procedures is small and can be reduced with appropriate use of infection-control precautions (9,10).

[References and footnotes omitted]

[Add. 122]

[Am. J. Public Health Feb. 1990, Vol. 80, No. 2]

HIV Seropositivity of Needles from Shooting Galleries in South Florida

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Abstract: Needle/syringe combinations were collected from three shooting galleries in South Florida and tested for the presence of antibodies to HIV-1. Fifteen of 148 needles (10.1 percent) tested positive for HIV-1 antibody. Seropositivity rates did not vary by the day of the week of collection, nor by shooting gallery from which they were collected. When the needle appeared to contain blood residue, 20.0 percent were positive versus 5.1 percent with no blood residue. These findings suggest that needles/syringes used in shooting galleries are likely to serve as reservoirs and/or vectors of transmission of the HIV-1 virus, and that although visual inspection of the needle/syringe may be useful in lessening the chance for transmission, even the visually "clean" needles may result in transmission of infection. (*Am J Public Health* 1990; 80:150-152.)

Introduction

The self-reported sharing of needles and syringes, particularly those belonging to shooting galleries, has been consistently associated with increased risk for HIV-1 infection among parenteral drug users.[1-3] A shooting gallery is a place where addicts go to rent syringes and needles ("works") and to inject drugs.

Shared drug injection equipment can transmit HIV-1 by parenteral transfusion when residual, contaminated blood remains in previously used syringes and needles and that equipment is reused by another person. Blood residue is often present in the syringe because of the aspiration of venous blood into the syringe.

No one has assessed the extent to which equipment owned by shooting galleries is positive for HIV-1 antibodies. Recently a study of needle-and-syringe exchange programs in Sydney, Australia determined that antibodies for HIV-1 were present in 3.1 percent of 1,544 needle and syringe combinations exchanged at two exchange centers.[4] Risk of exposure to HIV-1 probably is higher in shooting galleries where a wide variety of people rent the same works and needle and syringe combinations are routinely reconditioned to extend their useful life far beyond the average nine uses reported by intravenous users in Miami who reuse or share personal works.

In order to examine the potential for HIV-1 transmission through the use of injection equipment available in high-risk settings, needle/syringe combinations were collected from shooting galleries frequented by parenteral drug users in Miami, Florida and were tested for antibodies to HIV-1. Although this study measures antibody and not the presence of an infectious dose of virus, these data help explain the association between shooting galleries and HIV-1 infection.

Methods

Selection of Shooting Galleries

Three shooting galleries located in Miami, Florida were selected as the sites from which to collect needle/syringe combinations. These were among the most frequently mentioned galleries that were attended by parenteral drug users enrolled in coordinated investigations of the natural history of HIV-1 and the evaluation of risk behavior reduction programs. Seroprevalence* among users enrolled in these studies is approximately 36.1 percent for Blacks who were not Hispanic, 30.2 percent for Hispanics,** and 8.9% for Whites who were not Hispanic. The majority of persons who frequented these galleries were Blacks who were not Hispanic. Each gallery was located in a different inner-city area known for high rates of drug use.

Field observations at eight additional galleries indicate that the modes of operation at the study sites were typical for area

shooting galleries. Each was housed in the residence of the gallery operator. All three rented used works for $2 and had bleach or alcohol available for cleaning works, and none sold drugs. Heroin and/or cocaine, both available for purchase nearby, were predominant. Syringe and needle repair and cleaning occurred in these galleries but no observations were made specifically for these needles and syringes.

Needle/Syringe Collection

Access to the three galleries was gained through the efforts of a staff outreach worker who had established a variety of contacts with the intravenous drug using population. He was a follow-up worker who spent all of his time locating drug using participants of a longitudinal study. Because he was not an outreach worker in an HIV prevention program, the shooting gallery operators were less likely to feel pressure to manipulate the selection of syringes. Needle/syringe units that had been used in the shooting galleries during the previous 24-hour period were collected each morning by the follow-up worker during two one-week periods in 1988. In order to avoid possible bias by day of the week, no more than six units were collected per site per day. Whatever more were available, six were selected by the follow-up worker without regard to appearance or condition.

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*Seroprevalence is based upon data from three coordinated studies of HIV infection among intravenous drug users (N = 1364) in Miami, Florida.

**The study population includes Hispanics who are White and Hispanics who are Black; they are Hispanic in culture and do not identify themselves as White or Black.

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Each gallery operator was paid a flat fee of $24 per visit and did not participate in the selection of syringe units.

Each morning after collection, the needle and syringe combinations were immediately transported to the University of Miami and visually graded as follows:

1. "Visible Blood"--The needle/syringe appeared to contain liquid or dried blood.
2. "Dirty, No Visible Blood" -- The needle/syringe contained visible dirt, or stains, but no blood.
3. "Clean. No Visible Blood" -- The needle/syringe contained no visible dirt, stains or blood.

Needles were then delivered to the laboratory where they were tested for the presence of antibodies to HIV-1.

Laboratory Methods

A pilot study had been conducted with 17 needle and syringe combinations under conditions simulated to represent the condition of the needles/syringes which would be collected at the shooting galleries. Using the blood samples from a high-risk group whose blood was sent to the laboratory for routine testing, 200 µl of blood from each sample were drawn into a syringe and expelled. After standing overnight each needle/syringe was rinsed with 200 µl of saline and flushed 10 times with the saline solution. This solution was assayed twice by ELISA (Abbott Enzyme Immunoassay).

Ten samples from the test needle/syringe combinations were found to be reactive for HIV-1 antibodies confirmed by Western Blot and seven were non-reactive. These results were identical to the results of the routine testing for HIV-1 antibodies done on the blood samples.

Needle/syringe combinations collected from shooting galleries likewise were rinsed with 200 µl of saline, flushed 10 times and assayed in the same manner as the test needles. These procedures were similar to those used by Wolk[4] except that needles were tested more quickly and less saline was used in our study.

Needle/Syringes Not Tested

The field worker collected needle/syringe units regardless of condition or potential for testing or reuse. Sixty-two of the units collected could not be tested because of physical damage to the syringe (58 percent) or a clogged/damaged needle (48 percent). Needles and syringes were collected for three days during a subsequent week until 50 testable combinations had been received from each site. In two cases, results of HIV-1 testing were indeterminate on Western Blot, and were excluded from the analysis.

Analytical Procedure

Differences in proportions and 95 percent two-sided confidence intervals around the difference were calculated.

Results

Fifteen (10.1 percent) of the 148 needle/syringe combinations tested were found to be positive for HIV-1 (Site 1 = 10.2%; Site 2 = 12.3%, Site 3 = 8%) and 133 were negative (89.9 percent). There were no differences in seropositivity by day of the week on which needles were collected.

The relation between the graded condition of the needle/syringe combinations and the presence of HIV-1 antibodies

is presented in Tables 1 and 2. Twenty percent of the equipment combinations which had visible remnants of blood had evidence of HIV-1 antibodies, compared to 5.1 percent in combinations without blood. Thus the risk of having an infected needle/syringe was approximately four times higher if the needle/syringe appeared to be bloody. No differences in seropositivity were observed when syringe and needle combinations which contained no visible blood were partitioned into clean (4.7 percent) and dirty (5.5 percent) categories (Table 2).

Table 1 -- Seropositivity of Needle/syringe Combinations without and with Visible Blood

Appearance	Total Tested	Proportion HIV Ab+*
	N	%
No Visible Blood	98	5.1
Visible Blood	50	20.0
Total	148	10.1

*Difference in proportions 0.149 (0.06, 0.21)

Table 2 -- Seropositivity and Visual Appearance of Needle/Syringe Combinations without Visible Blood

Appearance	Total Tested	Proportion HIV Ab+*
	N	%
Clean	55	5.5
Dirty	43	4.7
Total	98	5.1

*Difference in proportions 0.008 (-0.06, 0.12)

Discussion

This study confirms epidemiological evidence that the use of needles belonging to shooting galleries can transmit HIV-1. Ten percent of the needle-syringe combinations which were tested from three shooting galleries were positive for antibodies to HIV-1. The use of such needles presents a clear public health hazard.

Shooting galleries provide needle and syringe combinations for "rent" to drug users. The combinations usually are held by the gallery operator or laid out on a table and are selected for use by either the operator or customer depending on house rules. Water and in some cases, alcohol or bleach, are available to rinse the works, i.e., to clean the syringe and needle. The custom in many galleries is to clean the works after injecting--not prior to injecting. However, there are no mechanisms to assure that works are cleaned after use and in many instances we found they were not cleaned. Moreover cleaning often entailed simply rinsing with water rather than bleach or alcohol.

The data from this study suggest that the choice of clean-appearing injection equipment does not eliminate the possibility of using an HIV-1 infected needle. Needle and syringe combinations which contain visible blood were more likely to be positive for antibodies to HIV-1 than those that do not contain visible blood, but the selection of a needle without visible blood was no assurance of safety. This is important because drug users may mistakenly believe that they are capable of determining which used works are "clean" i.e., safe.

Works rented in most shooting galleries are returned for future use until they are no longer functional. In this study, 29.2 percent of the collected needle/syringe combinations were clogged and could not be tested. No attempt was made to test these non-functional needle/syringe combinations because of the risk of a needle stick or contamination by

laboratory personnel (who would have had to remove needles to test the contents of the clogged needle or syringe). However, 60 percent of the untested combinations contained blood residue while only 33 percent of those tested contained visible blood. Thus, our estimate of seropositive needle-syringe combinations probably is a

conservative estimate of seropositivity in usable needle/syringe combinations which at the time they were collected were available for use by shooting gallery customers.

Our choice of shooting galleries was not by random sample and does not purport to represent all shooting galleries in the area. For obvious reasons, it is not possible to draw up an inclusive list of shooting galleries from which to randomly select representative galleries. However, those studied were three of the largest and most frequented galleries in the South Florida area. The operators of each gallery reported they serviced an average of 125 intravenous drug users per week and many of these clients rented works on more than one occasion.

As with other drug use issues, the solution to the problem posed by HIV-1 contaminated needles and syringes is not simple. Intravenous drug users frequent shooting galleries for a variety of reasons.[5-6] In communities such as Miami where new syringes may be purchased by prescription only and possession of drug paraphernalia is illegal, galleries provide the user with a place to inject which is relatively safe from police intervention. Shooting galleries increase the availability of needle and syringe combinations and some also sell drugs. Galleries provide a place where drug users can socialize with other drug users. At some locations prostitution is common. Even faced with evidence that shooting galleries are sources of HIV-1 infection, many users still frequent galleries.

Intervention programs must be developed to reach intravenous users who continue to use shooting galleries. Both the users and gallery operators need to be educated on the necessity of either using new works or cleaning works and need to be taught proper cleaning techniques. Gallery operators exercise control over injection behavior at their locations. If they become convinced that sterile needles and syringes are essential, those central figures could have considerable influence upon a large number of other users. For example, two gallery operators in Miami have been observed requiring all customers to purchase new works.

If risk reduction is to occur among users who continue to inject drugs, techniques to clean works must be incorporated into the value system of the operators of the galleries as well as the individual parenteral drug users and made a part of the shooting behavior of those users who frequent shooting galleries.

[acknowledgments, references and advertisement omitted]

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[HIV - Advances in Research and Therapy October 1992, Volume 2, Number 3]

QUANTITATION OF HIV-1 IN VIVO

David D. HO., MD

[footnotes omitted]

Over the last 8 years, molecular biologists have diligently studied the genome of human immunodeficiency virus type 1, mapping its genes, analyzing their function, and unraveling their complex interactions. Because of intense research by many scientists using powerful new technology, more has been learned about the genetic virtuosity of HIV-1 in a shorter time than about any other human pathogen.

What has not been learned about HIV-1 is equally impressive. Although its complete nucleotide sequence has been determined and catalogued, we still know little about where HIV-1 originated, how it interacts with the human immune system, and why it eventually overwhelms the body's defenses. The clinical virology if HIV-1 has lagged behind molecular analysis of the virus.

In recent years, some of my research has attempted to improve our understanding of the behavior of this virus in the living host. With colleagues at Harvard Medical School, now the Aaron Diamond AIDS Research Center and New York University School of Medicine, I have focused on identifying and quantitating HIV-1 in patients, from the first weeks of acute infection through the final stages of AIDS. What we have learned has changed our perception of the size of the viral burden in patients and of how the viral load itself affects the pathogenesis, transmission, and treatment of the disease.

For several years after the discovery of HIV-1 in 1983, it was widely assumed that the expression of the virus in infected patients was low, perhaps because the expression of the first identified human retrovirus, human T lymphotropic virus type I (HTLV-I), was known to be low. This belief was also based

partially on an early study that showed a low actual rate of isolation of HIV-1 from infected persons and on an in situ hybridization study that found that HIV-1 messenger RNA was expressed in only 1 of 10,000 peripheral blood mononuclear cells (PBMC). This evidence seemed so compelling that there was even debate over whether HIV-1 was actually the cause of AIDS.

Yet, no study in the first 5 years of HIV research had systematically quantitated HIV-1 in vivio. To fill this gap in our understanding of how HIV-1 actually behaves in humans, we undertook such a study in 54 adults - 53 men and 1 woman - who were seropositive for HIV-1 but were not receiving antiviral therapy, although 18 had taken zidovudine earlier. Twenty of these patients had AIDS, 18 had AIDS-related complex (ARC), and 16 had no symptoms of their infection. The CD4-positive lymphocyte counts reflected the different degrees of morbidity in these three groups. Among the AIDS patients, the mean CD4 count was 88 lymphocytes/mm3; in the ARC patients, the mean was 147 lymphocytes/mm; and in the asymptomatic patients, it was 462 lymphocytes/mm.

The study also included 20 patients with AIDS or ARC who received 400 to 1200 mg of zidovudine daily for at least 4 weeks and 7 other AIDS or ARC patients who were evaluated before and after 4 weeks of treatment with 800 to 1200 mg of zidovudine daily. Twenty-two seronegative gay men were evaluated as controls.

The goal of the study was to quantitate HIV-1 in PBMC (that is, lymphocytes and monocytes) and plasma by end-point—dilution cultures. In a study completed with Harvard colleagues in 1985, we were able to detect HIV-1 in monocytes from peripheral blood of infected patients – two with ARC and two still asymptomatic. We speculated then that monocytes served both as a major reservoir of infection and as a means for HIV-1 to infect the central nervous system, lungs, and other tissues, where the virus could be expressed when the monocytes matured to macrophages.

Even earlier, the isolation in our laboratory of HIV-1 from the semen and blood of gay men who showed no symptoms

confirmed the suspicion of a carrier state in infected individuals who appeared to be healthy. Still, we had no information about the actual quantity of HIV in the blood of such patients.

The end-point—dilution method used in our quantitation study consists of culturing serial dilutions of plasma and PBMC. Cultures were considered positive if the concentration of p24 antigen in the culture supernatant exceed 1000 pg/mL on a single determination or 200 pg/mL on two or more determinations (p24 is a protein in the core of the HIV-1viron). The end point that gives this quantitative method its name is the lowest number of PBMC or the smallest volume of plasma that will produce positive cultures. This number is expressed as tissue-culture-infective doses (TCID) per 10 PBMC or as TCID per milliliter of plasma.

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[Figure 1 omitted]

Our results were surprising, given the findings of previous studies. Not only was HIV-1 recovered from the plasma and PBMC of every HIV-1 positive patient (meaning that the sensitivity of the test was 100%), but the levels of virus in PBMC and plasma were much higher than had been estimated earlier (Figure 1 [omitted]). The failure to detect HIV-1 in seronegative controls verified that the specificity of the culture method was 100% and that there was no evidence of contamination of cultures by other viruses in the lab.

Figure 1 also shows that the viral titers of both plasma and PBMC were considerably higher in ARC and AIDS patients than in infected individuals who were asymptomatic. The mean HIV-1 titers in plasma were 30 TCID/mL in asymptomatic patients, compared with 3,200 TCID/mL in patients with ARC and 3,500 TCID/mL in those with AIDS. To express these results in another way, in asymptomatic patients, the level of

HIV-1 was approximately 1 TCID per 30 μ.L of plasma, whereas in symptomatic patients, it was 3 TCID per 1 μ.L of plasma. Circulating antibodies in individuals infected with HIV-1 clearly do not neutralize the virus.

In infected patients with no symptoms, HIV-1 was detected in 1 of every 50,000 PBMC, compared with 1in every 400 PBMC in patients with ARC or AIDS. If one assumes that the primary reservoir of HIV-1 is CD4-positive lymphocytes and that the CD4-positive lymphocytes in these patients average 5%, then 1 in 20 CD4-positive lymphocytes in symptomatic individuals is infected by the virus. How many of these cells actively express the virus is not clear. If earlier in situ hybridization studies are reliable, less than half of 1% of infected PBMC may express HIV-1.

The many implications of this study involve the pathogenesis, transmission, and treatment of HIV-1 infection. The large viral burden found in these patients could not have been predicted on the basis of earlier studies. It suggests that the cytopathic effects of HIV-1 alone, rather than indirect immunologic mechanisms, may be responsible for the onset of immune dysfunction. The high levels of HIV-1 in plasma indicate that circulating neutralizing antibodies cannot clear the retrovirus or even inhibit its proliferation, as they do in other viral infections. It seems clear that circulating antibodies are not the primary determinant in a patient's progression from acute to chronic to full-blown infection.

All of the HIV-1-infected patients in this study who were not receiving

TABLE. Calculation of levels of HIV-1 potentially transmitted from blood of HIV-1-positive patients

	Tissue-culture-infective doses (TCID) of HIV-1	
	From asymptomatic HIV-1-positive patients	From patients with ARC or AIDS
250-mL unit of blood for transfusion	15,000	1,750,000
1 to 10 mL of blood for laboratory evaluation	60 for 1 mL 600for 10 mL	7,000 for 1 mL 70,000 for 10 mL
10 to 100 µL of blood on needles shared by intra-venous drug users	0.6 for 10 µL 6 for 100 µL	70 for 10 µL 700 for 100 µ
1 µL of blood in needlestick injuries	0.06	7

Figures are based on the calculation (derived from study data) that whole blood of asymptomatic patients harbors approximately 60 TCID/mL and that blood from ARC or AIDS patients carries 7,000 TCID/mL

ARC, AIDS-related complex; AIDS, acquired immunodeficiency syndrome.

Antiretroviral therapy had detectable virus in their plasma. This finding indicated that HIV-1continues replicating even in patients with no detectable p24 antigen in their serum. These results strongly suggest that there is no truly latent phase of HIV-1 infection. A corollary is that serum p24 antigen is not a consistently reliable marker of disease progression.

David Baltimore and Mark Feinberg have suggested that our end-stage—dilution technique of measuring plasma viremia may become a better marker than those now in use (serum p24 levels, CD4 cell counts, and nonquantitative HIV-1 cultures) and may help speed identification of effective antiviral agents. Indeed, this quantitative technique accurately reflected the effects of zidovudine in those patients in our study who were taking the drug. HIV-1 titers fell significantly in the plasma of the seven patients who received zidovudine (AZT) for 4 weeks, although levels of HIV-1 did not fall consistently in the PBMC of these patients. Quantitative culture techniques have also proved reliable in calculating the effects of didanosine (ddI) in reducing plasma titers of HIV-1. On the other hand, these same techniques have demonstrated that soluble CD4 and trichosanthin (GLQ223) did not lower titers of HIV-1 in plasma (Daar ES, Mohri H, Ho DD. 1991. Unpublished data). Quantitative polymerase chain reaction (PCR) technology has also been used to determine the anti-HIV-1 activity of different therapeutic agents. Data from these studies suggest that quantitative culturing of HIV-1 and quantitative PCR should prove useful in monitoring the invivo efficacy of drugs, as well as in determining clinical cofactors that can result in enhanced replication of HIV-1.

These findings also deepen our understanding of the transmission of HIV-1 (Table). On the basis of our results in the 54 infected patients in this study, we calculate that each milliliter of whole blood of asymptomatic patients harbors approximately 60 TCID of HIV-1 and that each milliliter of whole blood of ARC or AIDS patients contains approximately 7,000 TCID. Thus a 250-mL unit of blood from an asymptomatic patient could contain 15,000 TCID of HIV-1, while a 250-mL unit from a symptomatic patient could harbor as much as 1,750,000 TCID. These estimates clearly underline the need for careful screening of the blood supply.

Hospital laboratory workers who routinely handle 1 to 10 mL of blood may thereby expose themselves to 7,000 to 70,000 TCID of HIV-1 if the blood is from a patient with AIDS. Thus,

it is imperative that recommended precautions in handling blood samples should be resolutely observed. The risk of transmission

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from needlestick accidents is proportionately lower but still exists. If the amount of blood involved in such accidents is 1 μL, a health care worker would risk exposure to 0.06 TCID with blood from an asymptomatic patient and 7 TCID with blood from a patient with AIDS. More blood is exchanged when intravenous drug users share needles, perhaps 10 to 100 μL. An asymptomatic person might transmit 0.6 to 6 TCID under those circumstances, whereas an AIDS patient would transmit 70 to 700 TCID.

The therapeutic implications of our findings derive from our evaluation of the antiretroviral agent zidovudine in this study and from our conclusion that there is no totally latent phase in the course of HIV-1 infection. The titers of HIV-1 in plasma decreased from a mean of 1,700 TCID/mL to 100 TCID/mL—a 94% reduction—in the seven patients treated with 800 to 1200 mg of zidovudine daily for 4 weeks. Furthermore, plasma levels of HIV-1 were considerably lower in the 20 patients who received long-term antiviral therapy. We failed to detect HIV-1 in the plasma of only four seropositive patients in this study, and all four were receiving zidovudine.

It is important to note that zidovudine did not completely suppress HIV-1. Specifically, it had no consistent effect on titers of HIV-1 in PBMC. The HIV-1 titers in PBMC may not drop quickly upon treatment with zidovudine because the virus may be replicating slowly or not at all in many of the cells and thus may be refractory to the effect of the drug on reverse transcription. The titer in PBMC may decrease only after a prolonged course of drug treatment.

Our conclusion that there is no latent period—that replication of HIV-1 is continuous—is one argument for starting antiretroviral therapy earlier in the course of infection. In an editorial accompanying the publication of our findings, David Baltimore and Mark Feinberg reasoned that the observed correlation between increasing viral burden and disease progression argues for earlier anti-HIV-1 therapy. In addition, they noted, our finding that plasma viremia is a better indicator of clinical status than p24 antigen levels suggests that the reemergence of higher levels of HIV-1 replication can be predicted earlier and postponed longer if less toxic therapy can inhibit this replication. Baltimore and Feinberg concluded that decreasing viral replication through antiretroviral therapy may also decrease transmission of HIV-1.

Beginning antiretroviral therapy earlier may have its own risks. Our finding of high titers of HIV-1 may help explain the emergence of resistance of the virus to agents such as zidovudine. As replication of HIV-1 increases in infected patients, so may the risk that mutant strains resistant to particular antiviral agents will emerge. Beginning therapy earlier may only hasten the development of resistance.

Although our work had yielded estimates of the viral burden in three groups of patients—those who are asymptomatic, those with ARC, and those with AIDS—similar studies had not been done in patients who had only recently become infected. These patients' symptoms—most typically fever, lymphadenopathy, pharyngitis, myalgia, gastrointestinal distress, and rashes—generally resolve within 1 to 2 weeks as antibodies to HIV-1 develop.

In 1985, my colleagues and I published two studies in which we reported isolating HIV_1 from patients during this initial acute reaction to infection. Three of these patients had the typical symptom complex, and two had acute, self-limited meningitis. However, we did not determine the levels of HIV-1 in these patients.

Is the viral burden during this acute stage of infection as great as we found it to be in patients with ARC or AIDS? In

fact, it is often much greater. We reached this conclusion in a study of four gay men whose HIV-1 titers were quantitated during the acute illness that followed their infection with the virus. Again we used end-point—dilution cultures to calculate the TCID in plasma and PBMC, and we confirmed the results with a PCR technique to count the copies of viral DNA. As initially high titers of HIV-1 dropped spontaneously in all four patients, we were also able to confirm seroconversion by testing sequential plasma samples for HIV-1- specific antibodies by enzyme immunoassay and western blot assay.

In plasma, the peak titers of HIV-1 were 10,000 TCID/mL in two patients during the acute seroconversion phase of the illness and only slightly lower in a third. In the fourth patient, the level of HIV-1 plasma was 1,000 TCID/mL. Over a period ranging from 9 to 39 days, the levels of cell-free virus in these four patients plummeted to 1 to 2 TCID/mL, lower than the mean plasma titer of 30 TCID/mL found in asymptomatic

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[Figure 2 omitted]

infected patients in our earlier study.

In PBMC, the peak titers measured 10,000 TCID/10 cells in the same three patients who had high plasma titers and 100 TCID/10 cells in the single patient who had the lower plasma titer. These high levels also dropped spontaneously in 10 to 36 days—with no antiretroviral therapy—to 1 to 10 TCID/10 cells. And again, these titers in PBMC were lower than the mean of 20 TCID/10 found in our earlier study.

Although the titers of HIV-1 in all four of these patients can be considered high compared with the titers in asymptomatic infected patients, levels of the virus were much lower in one of these patients than in the other three. This patient's presenting symptoms were distinct from those of the other patients: he had an unusual, life-threatening Guillain-Barre syndrome in addition

to more typical "flulike" symptoms. Still, there is no simple explanation for the difference between his levels of HIV-1 and those of the other patients. It is possible that patients with fewer symptoms than these four in the weeks after infection with HIV-1may have even lower levels of circulating virus. Why the symptoms of HIV-1—infected patients are so variable at the time of seroconversion is also unclear. Possible contributing factors include the patient's immune response and the quantity, virulence, or cellular tropism of the particular

infecting strain of HIV-1.

Some authorities speculate that aggressive treatment of this acute infection –when it can be identified—may block viral replication and delay the later onset of infection. Carefully controlled studies should be designed to determine whether early therapy may have such effects.

Other results of this study are less equivocal. First, the viral load in just-infected patients is often as high or higher than the viral burden of patients in the final stages of infection—those with ARC or AIDS. Second, there is at least a brief period when these individuals are seronegative yet highly infective. Third, these patients appear to mount a rapidly effective immune defense against the infection. It is conceivable that the transient viremia in these patients may reflect a lack of stimulated target cells in the human host. We believe it is more likely that the steep decline in circulating virus is the result of a potent immune response, possibly involving cytotoxic T lymphocytes. These killer cells effectively limit infections by other viruses, such as influenza and lymphocytic choriomeningitis viruses, and they are strongly activated throughout the chronic stage of HIV-1 infection.

Why this response eventually deteriorates remains a seminal question of AIDS research. Perhaps a better

understanding of this or other immune mechanisms responsible for the initial control of infection will eventually help forestall the onset of AIDS.

[References Omitted]

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HIV

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Failure of Gloves and Other Protective Devices to Prevent Transmission of Hepatitis B Virus to Oral Surgeons

(Defendant's Exhibit 3)

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Arthur L. Reingold, MD; Mark A. Kane, MD, MPH; Allen W. Hightower, MS

A survey of 434 oral surgeons was conducted to examine risk factors for hepatitis B virus (HBV) infection. Overall, 112 (26%) of the participants demonstrated serologic evidence of past or current infection with HBV. Seropositivity was significantly associated with age, number of years in practice, and year of graduation from dental school but not with other variables examined, such as the number of patients seen annually or the number of patients seen who were at high risk of HBV infection. The strong correlation between years in practice and seropositivity was unaffected by reported use of gloves, face masks, or eye shields. The use of gloves and other protective devices does not appear to offer substantial protection against HBV exposure in oral surgeons, and all oral surgeons should receive HBV vaccine. (JAMA 1988:259-25588-2560)

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Report requests to Hepatitis Branch, Division of Viral Diseases, Center for Infectious Diseases, Centers for Disease Control, Atlanta, GA, 30333 (Dr. Kane).

[Footnotes omitted]

AN INCREASED risk of exposure to and infection with hepatitis B virus (HBV) has been documented among health care providers and laboratory personnel, including surgeons, pathologists, oral surgeons, dentists, renal dialysis unit employees, blood bank technicians, and clinical laboratory technicians. Seropositivity rates among oral surgeons have been reported to be as high as 30%, with 1% to 2% of practitioners actively infected at any one time. A direct correlation between age and/or years in practice and seropositivity has been observed among health care workers, indicating that they are at continuing risk of infection throughout their careers.

Studies in renal dialysis units have documented that the routine use of gloves in conjunction with other infection-control procedures can substantially reduce the excess risk of HBV infection among health care workers. It has been suggested that routine gloving by dentists and oral surgeons might reduce their risk of exposure to HBV. Two studies have examined whether gloving decreases HBV seropositivity among dental professionals, but neither study had a sufficient number of participants who wore gloves to permit statistical analysis. Because the efficacy of using gloves and other protective devices in preventing HBV infections in this setting remains undetermined, we studied this question using data collected before the use of the HBV vaccine became routine.

METHODS

A serosurvey was conducted at the 63rd annual meeting of the American Association of Oral and Maxillofacial Surgeons (AAOMS) in Washington, DC, between Sept. 18 and 22, 1981. All AAOMS members were notified by mail about the study before the convention. All participants gave written informed consent, had a blood sample taken, and completed a questionnaire. The following information was obtained: age, sex, and race of the practitioner; year of graduation from dental school; years in practice; state of residence; type of practice; number of patients seen annually; geographic origin and socioeconomic and racial characteristics of their patient population; estimated number of drug addicts, institutionalized mentally retarded persons, renal dialysis patients, male homosexuals, and Southeast Asian refugees among their patients; and use of gloves, masks, and eye protection (including eyeglasses) in their office practices. Information about the use of disinfectants, intravenous therapy, etc, has been reported separately.

Laboratory Procedures

Hepatitis B surface antigen (HBsAg) was tested for by radioimmunoassay (Ausria II, Abbott Laboratories, North Chicago, Ill); positive specimens were retested after neutralization with hightitered goat antibody to HBsAg. Antibody to HBsAg and antibody to hepatitis B core antigen (HBcAb) were tested for by radioimmunoassay (Ausab and Corab, respectively, Abbott Laboratories). Testing was performed at the Centers for Disease Control, Atlanta.

Statistical Methods

Data were analyzed in univariate fashion using a two-tailed t test or χ^2 test, as appropriate. Data were analyzed in multivariate fashion using unconditional logistic regression with a model that included all the above variables.

RESULTS

Of the 1628 AAOMS members who attended the meeting, 434 (27%) participated. Of the 434, 112 (26%) were seropositive (ie, had evidence of current or previous HBV infection), including nine (2%) positive for HBsAg, 99 (23%) positive for antibody to HBsAg, and four (1%) positive only for HBcAb. Thirteen HBcAb-negative individuals with low-level reactivity for antibody to HBsAg (<10 sample ratio units) were seronegative.

The sex and race of the oral surgeon could not be evaluated as risk factors for seropositivity because virtually all of the participants were male (99%) and non-Hispanic whites (96%). Of the variables examined, only three were significantly associated with seropositivity in univariate analyses: age, years in practice, and year of graduation from dental school. Seropositive oral surgeons were significantly older (mean age, 46 vs 41 years, $P<<.01$ by : test). When examined using logistic regression, the seropositivity rate increased significantly with increasing age ($P<<.01$ x test for linear trend). None of the other variables examined, such as the number of patients seen annually (mean, 2592 for seronegative practitioners and 2603 for seropositive practitioners), demographic characteristics of the patients treated, or estimated number of patients treated who were at high risk of

HBV infection, were significantly associated with seropositivity in univariate analyses or in the multivariate analysis.

Table 1. –Glove Use by Oral Surgeons Reporting Unchanged Gloving Practices Throughout Their Professional Careers

Patient Encounters in Which Gloves were Worn, %	No. of Oral Surgeons	No. (%) Seropositive
0	30	9 (30)
1-10	107	24 (22)
11-89	23	3 (13)
90-99	14	4 (29)
100	45	7 (16)
Total	219	47 (21)

[Table 2 omitted] [Chart omitted]

To examine whether the use of gloves, face masks, or eye shields in the office protected against HBV infection, we looked at only those oral surgeons who reported following the same usage pattern for all their years in practice. For example, 219 oral surgeons reported the same frequency of gloving throughout their professional careers; 137 (63%) reported using gloves during 10% or fewer of their patient encounters, and only 45 (21%) reported using gloves during all patient encounters (Table. 1). The use of gloves in the office could not be demonstrated to have a protective effect against HBV infection, in that wearing gloves during a higher proportion of patient encounters was not associated with a lower risk of HBV infection. Also, the same increase in seropositivity with increasing age or years in practice was seen among oral surgeons who never or almost never used gloves (<10% of patient encounters) and those who used gloves for all or almost all patient contacts (>90% of patient encounters) [Figure omitted]. Limiting the comparison to oral surgeons who never used gloves and those who always used gloves produced similar results, although the total number of individuals in these two categories (30 and 45, respectively) was small. In similar analyses, more

frequent use of face masks and eye shields was not associated with a lower rate of seropositivity.

We also examined current use of gloves, face masks, and eye shields by oral surgeons and the relationship between such use and age. At the time of the survey, 105 (24%) of the 431 oral surgeons who responded to the question were using gloves during all patient contacts, while 236 (55%) were using gloves during 10% or fewer of patient contacts. Current glove use also did not affect the rate of seropositivity (seropositivity rates were 32%, 25%, 24%, 28% and 23% for oral surgeons who used gloves with 0%, 1% to 10%, 11% to 89%, 90% to 99%, and 100% of patients, respectively; not significant, χ test for linear trend).

Use of face masks was uncommon. Only 25 (6%) of the practitioners used face masks 100% of the time; 39% wore eye protection <10% of the time), probably because regular eyeglasses were included in this category.

Current gloving practices were inversely related to age. Thus, routine use of gloves was significantly more frequent among younger practitioners than among older practitioners (Table 2). No relationship was observed between use of face masks and the practitioner's age, while use of eye protection was directly related to age, again, probably because regular eyeglasses were included in this category.

COMMENT

Our results, showing a strong correlation between seropositivity and age or years in practice, agree with previous findings. Similarly, the lack of an effect of the number of patients treated annually on seropositivity has been noted previously. If the increase in the rate of seropositivity associated with increasing number of years in practice were due solely to the cumulative effect of being exposed to an increasing number of patients, one would expect seropositivity to correlate with number of patients treated. This discrepancy is most likely explained by the fact that the number of patients treated annually

at the time of the survey is not a good predictor of the total number of patients seen over the course of a professional career.

Seropositivity also was not associated with the number of "high-risk" patients

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[Add. 135]

(e.g, intravenous drug users, institutionalized mentally retarded persons, male homosexuals, and Southeast Asian refugees) treated. While it can be argued that many practitioners would not have accurate information as to how many of their patients fit into some of these categories, it is also likely that many of their exposures to HBV are from patients who are not in any identifiable high-risk group. A previous study has shown that even an extensive medical history is only 50% sensitive in identifying HBs-Ag-positive dental patients. If practitioners are more careful to take precautions when working with patients with identifiable risk factors for HBV infection, it may be that many of the exposures that result in transmission of HBV to oral surgeons are from patients without such risk factors.

Our study failed to find any protective effect from the use of gloves, eye shields, or face masks by oral surgeons. Although previous studies also have found no decrease in seropositivity rates among dental professionals who wear gloves, these studies included an insufficient number of glove-wearers to allow reasonable statistical analysis of the data. In our study, when we compared the seropositivity rate for practitioners who used gloves during 10% or fewer of patient contacts with the rate for those who used gloves during 90% or more of patient contacts and looked only at oral surgeons whose gloving habits had been the same throughout their years in practice, we found no difference. Based on the number of participants in each group, we would have had a greater than 90% chance of finding a statistically significant difference between these two groups if gloving 90% of the time or more were 75% effective in preventing the transmission of HBV. It is therefore unlikely that

we failed to detect a protective effect of this magnitude or greater. We had, however, only a 50% chance of finding that gloving 90% of the time or more is 50% effective. Furthermore, we had an insufficient number of participants to exclude the possibility that gloving 100% of the time offers substantial protection.

Given our low participation rate (27%) among convention attendees as well as the possibility that oral surgeons who attend a national convention may differ from those who do not, it is important to consider the possible effect of selection bias on our findings. If practitioners with a history of hepatitis and/or those who knew they were seropositive for HBV were more or less inclined to participate in the survey, our data would overestimate or underestimate the prevalence of seropositivity among oral surgeons. Similarly, if practitioners who use gloves or those who do not were more or less likely to participate, we would have overestimated or underestimated the prevalence of gloving. However, neither of these forms of selection bias could account for our finding that seropositivity increases in a similar fashion among those who use gloves and those who do not. It is true that if the combination of gloving practices and seropositivity status or history of hepatitis affected the likelihood of participation, we might have underestimated or overestimated the rate of seropositivity among glove-wearers or non-glove-wearers. Selection bias of this kind seems unlikely, given that prospective participants did not know that gloving was being examined in this study. More importantly, bias of this kind could not have produced the clear trend of increasing seropositivity with increasing age and increasing years in practice among glove-wearers, with a slope indistinguishable from that observed among non-glove-wearers.

It perhaps would not be surprising that wearing gloves might have only a minimal impact on the rate of exposure to HBV among oral surgeons. Of all medical and dental health care providers, oral surgeons are among those at highest risk, both because of the high proportion of patient encounters in which they are exposed to blood and because of the frequency with which they suffer puncture wounds and lacerations from sharp

instruments, broken teeth, and bone fragments. While gloves would be expected to decrease the frequency of patient and oral surgeon exposure to each other's blood, they offer only limited protection against puncture wounds and lacerations. Furthermore, it is plausible, although not documented, that patients requiring oral surgery might be more likely to be infected with HBV than are persons in the general population. It is, therefore, possible that our results are not applicable to other dental professionals and that gloves are more effective in preventing transmission of HBV when the majority of procedures produce less trauma and bleeding.

We found that only 24% of oral surgeons were using gloves routinely at the time of the survey (1981). While younger practitioners were more likely to use gloves routinely than older practitioners, more recent data show no evidence of an overall trend of increasing glove use. A national telephone survey of practicing dentists and oral surgeons in 1986 demonstrated that only 15% of respondents used gloves for all procedures and that only 44% had been vaccinated against HBV. A recent survey of dentists in New York City from October 1985 through May 1987 showed that only 31% always used gloves and that only 44% had received HBV vaccine. While we were unable to demonstrate an impact of the use of protective devices on exposure to HBV among oral surgeons, we urge that all dental professionals routinely use such devices in accordance with current recommendations. In addition, all dentists and oral surgeons should receive HBV vaccine.

We thank Edgar Mitchell, Ph.D. of the American Dental Association, Chicago, and John A. Babett, DMD, and Richard Vanselow of the American Association of Oral and Maxillofacial Surgeons, Chicago, for assistance in conducting the study; we also thank all of the participating oral surgeons for their time, enthusiasm, and assistance.

[References Omitted]

[Add. 136]

[Quintessence Int'l, Volume 25, Number 3/1994]

GUEST EDITORIAL

But the horse has left the stable

Enid A. Neidle*

I am writing in response to Dr. John Hardie's recent guest editorial, "Are current infection control practices justified?" (*Quintessence International* 1993;24:683-686).

Dr. Hardie's words and sentiments resonate familiarly in my mind, because in the nearly 8 years I served as Director of Scientific Affairs at the American Dental Association, I heard from hundreds (perhaps thousands) of dentists about the "irrationality" of the developing concerns about the need for stringent infection control in the dental office. They claimed there was no objective evidence that dental procedures are associated with the transmission of infectious disease, that (to use Dr. Hardie's own words) "not one case of dental equipment-mediated cross infection has been confirmed in the literature. . . . there are no controlled studies to demonstrate that the wearing of gloves will prevent the spread of either HIV or HBV during dental treatment," and that the pathogenicity of oral microbes is very low.

Some, but not all, of this is true. Let's first look at hepatitis B virus (HBV). By 1975 it was known that the incidence of HBV in dentists was three times that of the general population, and five times greater in oral surgeons. Between the mid-1970s and 1987, there were nine outbreaks of HBV that were directly traced to HBV-infected dentists, and two of these patients died. Dr. Hardie states that "HBV is readily prevented by immunization." This statement is true if there is an effective response to the vaccine, but it is widely known that the success

of the vaccine is affected by age, genetic factors, site of injection, and size of dose. Most recipients are not tested after vaccination, and therefore are not likely to know whether they have achieved immunity against HBV.

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Editor's Note: The purpose of the Guest Editorial is to allow authors to present their opinions on controversial issues. The views expressed by the author do not necessarily reflect the views of *Quintessence International* or its editors.

Hepatitis C virus, which Dr. Hardie does not mention, has been implicated by some infectious disease experts as a potential problem for dentists who, it is claimed, have a higher incidence of markers for hepatitis C virus than is found in the general population.[1] Other researchers contest this finding (Siew C, personal communication).[2] Hepatitis C virus, like HBV, is transmitted by blood, sexual contact, and household contact.

The Centers for Disease Control (CDC), in 1987, classified saliva in dentistry as usually contaminated by blood and, therefore, implicated saliva in dentistry as a vector for transmission of all blood-borne viruses. This meant that saliva in dentistry was one of the body fluids for which CDC recommended universal precaution. The Occupational Safety and Health Administration (OSHA) adopted the CDC's definition of saliva in dentistry and classified it as a potentially infectious material. Furthermore, transmission of HBV through saliva presumably not contaminated by blood has been documented.

Against this background of documentation for the transmission of the hepatitis viruses through saliva, several other defining events occurred in the 1980s and 1990s. A dentist, working in the inner city of New York and using gloves only sporadically, probably acquired human immunodeficiency virus (HIV) infection occupationally. Currently, six dental workers,

including a dentist, are under investigation for possible occupational acquisition of their HIV infections. The most outstanding and perplexing event was the case in which a Florida dentist transmitted HIV infection to six patients, in a manner as yet unknown. The two prime theories for these transmissions are that the patients were infected either through direct contact with the dentist's blood or through indirect contact via a dental instrument. These theories provide potent reasons for wearing gloves during dental procedures and for rigorous sterilization of dental instruments, whether they be critical, semicritical, or noncritical.

In a recent article, Mandel[3] cites a variety of infectious hazards in the dental office, including herpes simplex virus, cytomegalovirus, respiratory infections (in-

cluding tuberculosis), syphilis, and legionellosis. To be sure, transmission of these agents to dental workers is rare, and certainly ordinary masks offer scant protection against tuberculosis, but the possibilities of infection in the dental office are real and the even modest advantages offered by standard infection control would seem prudent and sensible.

Under the apparently onerous pressure of OSHA, CDC, and emerging infection control guidelines, dentists have espoused the requirement for scientific rigor with singular fervor. They demand to know where is the evidence, where are the controlled studies, where are the documented cases. I submit there is also something to be said for intuition. Some things seem intuitively right, even though there may be no double-blind, cross-over, rigorously controlled study to validate them. It was intuitively right that physicians, delivering their patients of babies, abandon their frock coats and unwashed hands to prevent the highly common post-partum development of pueperal fever.

It is intuitively right that when the dentist places an instrument or her own hands into a patient's mouth that they be scrupulously clean and sterilized, if possible. It is intuitively right that, prior to an injection of a drug, the site be swabbed with alcohol or iodine, even though no published data exist to show that, in the absence of the disinfection-infection of the skin, there are horrible consequences. To demand incontrovertible evidence that a handpiece has caused someone's death or serious infection before willingly agreeing to sterilize that handpiece is to fall into the moral obtuseness of the cigarette industry, which, 30-some years after the first report of the Surgeon General on the relationship between smoking and cancer, claims that no solid evidence for a cause-and-effect relationship has ever been published.

The American dental Association's Division of Scientific Affairs, well before the Florida case of provider-to-patient HIV transmission, believed strongly that it must review its infection control guidelines in light of the growing epidemic of infectious disease and the increasing patient concern about the safety of the dental office. The Acer case and the subsequent papers by Lewis and colleagues (given blinding publicity by the media) about the "dangers" lurking in the dental hand-piece raised the level of anxiety exponentially. The ADA was not alone in its concerns, for at the same time, other agencies were doing the same thing. Ultimately, after the most extensive and careful consultation with experts, researchers, handpiece manufacturers and disinfectant manufacturers, the ADA, the Food and Drug Administration, the Federation Dentaire Internationale, and then the CDC came out with revised infection control guidelines that were based on solid intuition, some scientific research, extensive knowledge about and experience with HBV, the facts of the Acer case, and the understanding that new infectious agents could appear as suddenly and mysteriously as HIV. These infection control guidelines have been adopted or endorsed by virtually every health agency, dental school, and hospital dental service in the country.

No, Dr. Hardie, there is no single research paper, no single "smoking gun" that proves that the handpiece is a vector for transmission of the deadly disease, or that failure to wear gloves will have fatal consequences; but, yes, current infection control practices are justified. They provide assurance that the dental profession is willing to go to some modest lengths to ensure the health of patients and to assuage their fears.

There are still dentists, like Dr. Hardie, who protest the inconvenience of infection control, as currently recommended by virtually every health agency, association, or educational institution in the world, But it's too late. The stable door is open, the horse has left, and the patient now comes to the dental office with some very specific and well-informed expectations of how that office and its professionals will be managed. But every dentist is free to choose how he or she will practice (unless he or she is an employee and falls under OSHA's standard). And so, Dr. Hardie, you too are free to choose, as are, in fact, are your patients.

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[Add. 138]

American Dental Association **ADA**

211 East Chicago Avenue
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May 25, 1990

Dr E. J. Neiburger
Vice President
American Association of Forensic Dentists
1000 North Avenue
Waukegan, Illinois 60085

Dear Dr. Neiburger:

I realize that it has taken me a long time to respond to your letter of March 10; but I thought I would try to find some papers establishing the scientific basis for using gloves in medicine and dentistry.

I did not believe I would find anything of substance in the literature of this century except for reviews. I was correct. In fact, from what I could determine little work has been done that would be considered suitable from a scientific standpoint.

Basically the use of gloves is an extension of Lister's attempt to perform surgery aseptically. Many of his successors simply added various techniques and types of garb in an attempt to reduce the transmission of microorganisms.

Do these additional measures help reduce disease transmission in surgery? Probably, but it is difficult to say based on controlled studies. Do I think we should wear gloves, masks and protective eyewear? Yes, but I think that dentists should be able to use their clinical judgment when it is appropriate.

You have to remember, however, that the OSHA regulations are not written for dentists but for dentists' employees. It is probably reasoned that these employees are not qualified to make the same clinical judgment decisions that dentists are qualified to do. Therefore, the regulations do not allow for much flexibility.

I understand that you are upset over the imposition of using these barrier techniques, but I believe this is part of a trend that goes beyond dentistry. Society is demanding a risk free environment. Consequently, barrier techniques, hazard communications, and more complicated waste disposal measures will continue to be major public issues well into the next century.

[Add. 139]

Dr. Neiburger
May 25, 1990
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If you wish to discuss this further, please feel free to contact me again.

Sincerely,

/s/Kenneth H. Burrell

Kenneth H. Burrell, D.D.S., M.S.
Secretary
Council-on-Dental Therapeutics
KHB;jy
Enclosures

[Add. 140]

American Dental Association **ADA**

211 East Chicago Avenue
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July 17, 1990

Dr E. J. Neiburger
Vice President
American Association of Forensic Dentists
1000 North Avenue
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Dear Skip:

I thought I had responded to your original request. Perhaps you could argue that my answer was too vague or obtuse so let me try again.

Infection control barrier techniques evolved from the aseptic techniques proposed and practiced by Lister. Gloves were believed to help prevent disease transmission to the patient. Masks and gown came later. All of these measures are based on observations made by surgeons as they watched their patients heal following surgery. Post operative infections probably were reduced with each subsequent infection control measure used today. A good deal of these infection control measures are based on empiricism. That being the case, a scientific evaluation by the Council on Dental Therapeutics would not be possible since scientific studies showing effectiveness would be necessary.

I agree with you that more scientific information should be available. I hope someday there will be.

Sincerely,

/s/ Ken
Kenneth H. Burrell, D.D.S., M.S.
Secretary
Council on Dental Therapeutics

KHB:jy

[Add. 141]

[J. Can. Dent. Assoc., Vol. 57, No. 1 (Jan. 1991)]

Scientific

Hepatitis B and HIV infections in dental professionals: Effectiveness of infection control procedures

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[Footnotes omitted]

ABSTRACT

In a survey carried out in 1987 and 1988 in Vancouver British Columbia, 704 dental professionals filled out an anonymous questionnaire and submitted a blood sample. Of those not immunized, 11% had infection with Hepatitis B, with dentists having a rate of 18%, significantly higher than other dental workers. Two carriers of HBsAg were detected and no individuals showed evidence of HIV infection. There were no differences in infection rates by the specific infection control practices. Oriental dental-care workers had a significantly higher rate of infection than other races surveyed. In a multivariate model, race and the number of years in practice were the only significant factors in predicting infection. The use of gloves did not show a protective effect, although a threefold protection rate for gloves is the smallest difference that could be reliably detected. The immunization rate with Hepatitis B vaccine averaged 46%, with fewer dentists being immunized than gygienists. Seroconversion rates were lower in older individuals. Dental professionals had a high risk of Hepatitis B and had not been adequately immunized. Reliance on gloves for operator protection appears to be inadequate.

[Abstract in French omitted]

Introduction

The increased risk of infection with HB virus in dental professionals has been well documented. The recent and continuing epidemic of HIV infection has heightened the concerns of these professionals to the potential and real risks of infectious disease. This has resulted in reinforcement of the recommendations made for the protection of patients and staff by advisory and regulatory bodies, including the *Canadian dental Association*, the *American Dental Association*, many state/provincial licensing bodies, *Centers for Disease Control*, and the *Federal Centre for AIDS*.

Evaluation of infection control procedures has been limited. Some studies have indicated that gloves were protective against HB infection, while others have not. Another study indicated that HIV transmission to dental professionals may be possible due to their professional activities. This study reports the results of a two-year survey which investigated the risk factors for infection, the efficacy of infection control procedures, and the response to immunization in dental professionals in British Columbia.

Method

The survey was carried out on individuals attending the annual meeting of the College of Dental Surgeons of British Columbia, in Vancouver in May 1987 and repeated in May 1988. An anonymous volunteer sample was recruited, via announcements and an information booth to complete a questionnaire and to donate a blood sample. Anonymity was assured by the use of a unique code number known only to the volunteer. Both the serum samples and questionnaires used this code. The volunteers were able to obtain the results of the serologic testing by requesting the results for their code number, but the investigators were unable to identify or trace any individual.

Questionnaires were coded for both years of the study by one of the investigators and data were entered in the year in which they were obtained. Questionnaires were identical except that the one for 1988 requested information on participation in 1987.

The serologic tests performed were Hepatitis B surface antigen (HBsAg), antibody to core antigen (anti-HBc), antibody to surface antigen (anti-HBs) and antibody to human immunodeficiency virus (HIV). All assays were carried out with Abbott Laboratories' (Chicago, Illinois) test kits. All positive assays were confirmed with replicate determinations.

The Hepatitis B status for an individual was determined as follows:

1) Susceptible - All tests negative, not immunized.
2) Carrier - HBsAg positive.
3) Infected - HBsAg positive and/or anti-HBc positive in an individual who had never been immunized.
4) Seroconverted - An individual who had been immunized and was anti-HBs positive.
5) No seroconversion - An individual who had been immunized and was anti-HBs negative.

The Hepatitis B status was determined in a hierarchical fashion; if an individual had anti-HBc present, he/she was considered infected, whether or not Hepatitis B

vaccine had been received.

Data Analysis

Statistical tests were carried out using a Chi-square test or Fisher's exact test if any cell was less that 5 expected. The level of significance was set at $p \leq .05$. Multivariate modelling was done using logistic regression analysis.

Results

Seven hundred and forty-eight individuals registered for the British Columbia Dental Convention in 1987, and two hundred and eighty-five individuals volunteered to complete the survey - a response rate of 38%. In 1988, there were approximately 1600 attending – 419 volunteers for a response rate of approximately 26%. Eleven per cent volunteered for both years. The dual responses were not removed from the analysis as the infection control questions related to the last six months or last year. As not all individuals answered all questions, the rates were determined for respondents only.

There were 285 males and 410 females. The mean age of the males was 42 years, and females 32 years. As expected, the dental hygienists, assistants and others were younger and had fewer years in practice than the dentists: 9.6 years for dentists, 8.6 for hygienists, 7.7 years for assistants and 7.4 for others.

Two individuals in this same sample were positive for HIV antibodies by the ELISA technique. Neither showed positive results by the Western Blot technique. These results are interpreted as negative for infection with HIV.

Risk Factors for Infection

The following section consists only of those individuals who were susceptible to HBV and had never been immunized, or those who were infected with HBV. The two carriers of HBsAg were included in the infected group. Those who were immunized were excluded unless they were anti-HBc positive. There were 323 susceptible (never immunized) and 39 infected individuals, a rate of 11% (Table 1). There were six inconclusive results that were not included in the analysis of this section. Non-respondents to a question were excluded and therefore the denominators vary for each group.

In looking at the personal risk factors for the transmission of HBV or potentially HIV, three individuals had used drugs intravenously. None were infected. Twenty had received blood or blood products, four (20%) of whom were

infected. Six had intercourse with a homosexual or bisexual male; one (17%) was infected. With respect to multiplicity of sexual partners, 17 reported no sexual partners, 253 reported 1 partner only, 26 reported 2 partners, and 17 reported 3 or more. The percentage infected in each group was 19%, 11%, 10%, and 6% respectively. None of these personal risk factors was statistically significant by chi-square from those not infected.

The racial background of this group was 317 Caucasian, 36 Oriental, and 10 others. Of those with an Oriental background, 31% had evidence of infection with HBV, while 9% of Caucasians and 10% of the others had evidence of infection. This was statistically significant: $p<0.001$.

The rates of infection by professional group were different. Dentists had an infection rate of 19%, hygienists 2%, assistants 4%, and others 6% (Table 1). The p value was <0.01. The number of years in practice was not significant for the rates of infection: 10% for those in practice 0-4 years, 4% for 5-9 years and 13% for greater than 9 years (Table II).

We assessed whether taking a medical history changed the rate of infection. Although 77% took a history and regularly updated it, the infection rates were similar to those who did not. Having members of the following risk groups in one's practice did not increase the rates of infection significantly: known HB carrier, high risk ethnic group, hemophiliacs, male homosexuals, or other high risk groups for HB carrier states.

The participants were asked whether they had had a penetrating wound in the past six months and the estimated number of such wounds. The infection rate in those with no wounds was 12/110 (11%), in those with 1-5 wounds 18/166 (11%), and for those with more than 5 wounds 3/33 (9%).

The use of gloves was rated: for all patients, 74%; all high-risk patients, 14%, occasionally, 9%; or never, 3%. The infection rate in all patients or all high risk group use was 34/303 (11%) and in those who used gloved occasionally or not at all, 3/42 (7%) (Table III). This difference was not statistically significant, $p=0.31$, one-tailed by Fisher's exact test. The odds ratio for the protective effect of gloves was 0.60, with test based limits, Miettinen method, of 0.18-2.06. The numbers in this

study were sufficient however only to detect an odds ratio of 3 for protection by gloves with a power of 80%. This small sample suggests that gloves do not decrease the risk of infection by more than three times but may still be protective at a lower level.

Similarly to gloves, the use of a mask or eye protection was not associated with an altered rate of infection.

Multivariate analysis using the unconditional logistic regression method was carried out to determine the strongest determinants for infection. The terms entered into the model included race, age, sex, years in practice, type of practice and use of gloves. Race and years of practice were significant terms in the model.

TABLE 1

Hepatitis B status in Dental Workers

	Susceptible	Infected	Carrier	Inconclusive	Rate
Dentists	124	30		2	19%
Hygienists	46	1		2	2%
Assistants	95	3	1	2	4%
Others	58	3	1	0	6%
Total	323	37	2	6	11%

TABLE II

Years of Dental Practice and Hepatitis B status

Years of Practice	Hepatitis B status			
	Susceptible	Infected	Carrier	Rate
0-4	74	7	1	10%
5-9	68	3		4%
10+	142	22		13%
Total	284	32	1	10%

Missing Observation: 122

Immunization

Immunization status was determined from the questionnaire. For those who reported immunization, seroconversion was determined from the anti-HBs result. Those who were reactive were considered seroconverted, those who were negative or inconclusive were classified as not seroconverted. Ninety-six percent of respondents stated that they were aware of the vaccine. In 1987, 113/278 (40.6%) were immunized, in 1988 this increased to only 208/413 (50%). Only 7.3% of individuals reported not being immunized because of having tested positive for HB markers. Knowledge of previous infection status was therefore not the reason for the low immunization rates. Of the dentists, 52%, of the hygienists 63%, of the assistants 33%, and of the others 18%, had been immunized. For those in practice 0-4 years, 50% were immunized, 5-9 years 57%, and ≥ 10 years, 50%.

The overall rate of seroconversion among those immunized was 68%, (Table IV). Dentists had a rate of 62%, hygienists 69%, assistants 84% and others 64%. In males the mean seroconversion rate was 62%, in females 73%. In males the seroconversion rate fell significantly with age (83% in 20-29 to 11% in the 60+). This was not found in females but there was only one individual in the 50-59 age group and no one in the 60+ range.

Discussion

This study recruited its population by a volunteer sample. Because of the nature of this recruitment, there is a strong subject selection bias operating which limits the conclusions made from the data. Because biases are non-random in nature, they cannot be controlled in the analysis. Areas with a particularly strong likelihood of a bias operating were identified.

Our study population included individuals who were at risk of HB or HIV infection due to independent factors, including racial origin, sexual exposures to homosexual or bisexual males, multiple heterosexual partners, intravenous drug

use and receipt of blood or blood products before 1986. Only Oriental racial origin was associated with increased risk. Since this exposure may occur before the occupational risk, it should be controlled in the study by adjusting the estimates of infection rates or alternatively in a multivariate model. Like others, we noted the infection rate increased with age and type of dental practice. In a cross-sectional study such as ours, we cannot tell whether the association with age represents a low rate of infection in each year, or a high rate of infection in the past that is no longer present, a cohort effect.

We used a multivariate technique, unconditional logistic regression, to determine whether race, years in practice, type of practice or sex was responsible for this increased risk. In this study, the most important determinant of risk was the number of years in practice. This presumably represents a marker for the number of potential exposures one may have had.

Although there are many reasons for taking a medical history from patients, we could not identify a difference in the risk of HB infection with taking a history or knowing that there were high risk patients in one's practice. This is likely due to the inaccuracies of a history in predicting risk.

The use of protection such as gloves, masks or eye protection did not decrease the risk of infection with HB virus, although a three-fold protection by gloves would have been required to be reliably detected by our study. Rheingold also failed to show protection from gloves. Our finding may have been due to a recent change in the use of protective measures, after infection had occurred, but Rheingold controlled for this factor. Other studies have found a protective effect of gloves, and they are recommended by advisory bodies.

Gloves were reported to be used in all cases by 74% of all practitioners. This change in behavior has occurred due to continued recommendations over the past several years. However, compliance to this recommendation is not universal. The findings of a recent study of a random sample of American dentists are relevant in discussing the findings of our study. That study found increased compliance with the use of barrier techniques; the use of gloves by general dentists increased from

23% in 1986 to 76% in 1988. It should be noted that more than 75% of dentists had been using gloves in the manner reported for two years or less. This change in pattern may not be demonstrable in their serologic status, due to the short time in which this change has occurred. Hygienists wore gloves 61% of the time in 1986, which was increased to 97% by 1988. Eyewear was worn in 72% of treatments in the 1986 survey and increased to 82% in the more recent survey. Masks were used for all patients by 26% in 1986 and increased to 47% in 1988.

In the same American survey, only 63% of hygienists, 52% of dentists, and 33% of auxiliaries were immunized in spite of repeated recommendations to do so. Similarly, we found that only 46% of participants in our study were immunized. An additional finding in this study was that seroconversion was 68%. This rate of seroconversion indicates the need for post immunization serologic study. The seroconversion rate for males decreased with increasing age.

TABLE III

Use of Gloves and Hepatitis B status: Protection from Gloves

	Hepatitis B status			
Glove Use	Susceptible	Infected	Carrier	Rate
Always	226	27	1	11%
High Risk	43	6		12%
Occasionally	29	2		6%
Never	10	1		9%
Total	308	36	1	11%

Missing Observations: 62

TABLE IV

Hepatitis B Immunization Status of Dental Workers

	Seroconverted	No seroconversion	Rate
Dentists	93	56	62%
Hygienists	59	27	69%
Assistants	42	8	84%
Others	7	4	64%
Total	201	95	68%

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[Add. 144]

Future studies should review the use of barriers (gloves) and the time for which they have been used. The number of years that these infection control measures have been in use may influence the findings in studies, as many of these measures have only been taken in recent years. The study by Epstein et al. evaluated patients prior to availability of HBV vaccine and was able to identify decreased infection rates associated with glove-use. This was prior to the general recommendations and the decreased pressure for infection control, and the more common use of these guidelines following the HIV epidemic. Those who reported compliance with infection control guidelines may have done so for a longer period on a regular basis. Gonzalez and Naleway, using data from the ADA annual meeting, showed that glove-use was associated with a decrease in HB infection and that this decrease was not explained by years of practice. The odds ratio of HB infection in those using gloves routinely compared to intermittently or never was 1.7 with test based limits of 0.95-3.42 (Miettinen). This is within the range found in our study. However, with an overall odds ratio of 1.7, the use of gloves for protection does not appear to be significantly effective. Both of our findings included no protection, (odds ratio + 1), within the confidence limits.

Other evidence for the use of gloves in infection control is present in studies of HBV carrier dentists who, upon

identification of their status, did not transmit infection to subsequent patients when gloves and other barriers were used. Another study demonstrated that the use of gloves, strict handwashing and limitation of use of intravascular devices was associated with a significant decrease in a hospital-wide outbreak of Staphylococcus aureus septicemia. These studies, however, indicate the effectiveness of gloves in protecting the patient and preventing person-to-person transmission rather than protecting the operator from puncture wounds. We would have detected a three-fold or greater protective effect in our study, indicating less protection than this for the dental professional. Gloves may be relatively more efficient at protecting the patient than the wearer. Moreover, the lack of HIV transmission in this study and the very low rate reported by Klein indicate a low risk of HIV transmission in dental practice, even if gloves may not efficiently protect from HBV infection. We did not find any evidence of a protective effect from masks or eye protection. This is consistent with the known routes of transmission of HB being sexual, percutaneous, and maternal-fetal. There is no evidence of droplet or airborne spread.

Only 46% of the participants in this study reported being immunized in spite of recommendations and the volunteer nature of the survey. Of these, only 68% had seroconverted with anti-HBs in spite of three or more doses of vaccine. The nature of the study, which likely encouraged individuals who were concerned about their immune status to volunteer, may have given a lower rate of seroconversion than expected. This rate is, however, not dissimilar to that reported by the Toronto General Hospital. With increasing age being a risk factor for lack of seroconversion, immunization as soon as possible after the increased risk of infection occurs, appears to be necessary. In the case of dental professionals, this would be on admission to the training facility and prior to patient contact. This becomes even more important since gloves did not protect from infection in our study population, and an infection rate of 11% is unacceptably high risk.

Conclusions

Hepatitis B infections continue to occur in dental professionals. Our survey indicates that the recent recommendations for the use of gloves and other infection control measures have either not had time to show a protective effect or do not completely protect the dental worker. While we do not conclude that gloves provide inadequate protection against acquiring Hepatitis B, we are concerned about the reliance on barrier methods as the sole method of protection. We strongly recommend the HB vaccine for dental professionals and stress that it be administered at as early an age as possible.

[Acknowledgments and References Omitted]

ADA
October 3, 1994

Volume 25, No. 18

Cost of OSHA compliance gets mixed response

Near the end of a four-hour presentation outlining the cost of infection control on dental practices, an insurer suggested that dentists had found a new "profit center."

Total infection control costs, amortized annually, surpass $5.4 billion for dental offices nationwide, a recent ADA survey found. Even after adjusting their fees, however, dentists still absorbed more than $500 million of that cost.

Put another way, total infection control costs went up on average $9.31 per patient visit while the average billing per patient visit increased $8.42.

Profit center? Though erroneous, the comment was indicative of the skepticism with which a few insurers received the survey finding during a presentation hosted by the ADA last month.

Most insurers, however, welcomed the Association's survey as an opportunity to obtain more accurate information so benefits plans can be adjusted to more accurately reflect the costs of care.

The survey, mailed to a random sample of 6,336 private-practice dentists throughout the United States, collected data for an 18-month period ending in January 1994.

Opening the survey presentation last month, ADA President James H. Gaines said, "I think all of us recognize the times we're living in as a period when tremendous changes are taking place and constant demands are being made upon our ability to adapt."

"Nothing is more important to our ability to adapt than our ability to put our hands on reliable information and the best data available," he stressed.

In search of data that would reveal whether infection control requirements were responsible for a 7 to 8 percent increase in dental fees between 1988 and 1990, the ADA decided two years ago to conduct a special survey.

"The purpose of the survey," said Dr. Gaines, "was to move beyond estimates and projections, and try to get a handle on the costs dentists actually encounter in complying with state and federal regulations on infection control."

Referring to the Occupational Safety and Health Administration's cost estimates for compliance with this standard, Dr. Gaines noted, "I suspect that none of us are all that surprised that OSHA underestimated costs."

"What is surprising and alarming," added the ADA president, "is the magnitude of the underestimate."

"Compliance with OSHA requirements alone is costing U.S. dental practices nearly 30 times more than what OSHA estimated," said Dr. Gaines.

OSHA estimated that compliance with its standard would cost dentists $87.4 million annually, or about $872 per practice per year.

OSHA compliance, according to the ADA survey, costs dental practices nationwide more than $2.7 billion annually, or nearly $24,000 per practice.

The total annual cost to dentists for all infection control measures - $5.4 billion - breaks down to a per-practice cost of about $45,718.

When OSHA calculated its cost estimate the federal agency "did not understand the dental office at all ... they came up with some fairly ridiculous numbers," said Donald House, Ph.D., president of RRC Inc., the dental-market consulting firm that assisted the ADA in developing the survey.

The ADA's "Survey of Infection Control and OSHA Compliance Costs" covered 10 categories: supplies, housekeeping, personal protective equipment/laundry, infection

control plan, vaccinations, training program, compliance, recordkeeping, sterilization of handpieces and exposure.

Dr. House, who presented the findings at the September gathering, stressed that the ADA data are consistent with statistics found in other surveys, such as the Quarterly Survey of Dental Practice, the annual Survey of Dental Practice and the Distribution of Dentists.

He noted, too, that all the figures in his presentation were averages for dental practices across the country. Actual figures were higher for some dental offices and lower for others.

Several questions, meanwhile, were designed to safeguard against exaggeration. Responses to these questions were compared to existing data from other surveys, noted Dr. House. When

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these comparative numbers didn't match, RRC eliminated the respondent from the survey results.

Other findings reported by Dr. House included the increase in time after the OSHA standard took effect in July 1992. The survey found that most dental staff positions spent twice as much time to disinfect environmental and work surfaces or change work surface and equipment coverings to meet OSHA requirements.

While dentists spent about 11 more minutes a day meeting the OSHA standard, dental office staff combined spent 79 more minutes a day to comply with OSHA.

And that isn't time dental offices can just squeeze into their existing schedules, Dr. House cautioned.

"There's a tendency to think dental personnel are sitting around doing nothing" - that dental offices should have no problem fitting another 79 minutes on infection control measures into the daily routine.

"That isn't the case," stressed Dr. House. Dentists have extended hours for their staff.

Commented Dr. Gaines, "I would be a very happy man if I could report to you that my colleagues and I are able to absorb

such costs - but economic realities being what they are, you would know that I was being less than honest."

"With dental office overhead expenses that traditionally account for about 65 percent of gross receipts," he added, "dentists cannot be expected to absorb the level of added costs outlined for you today."

Yet, according to the survey, that is precisely how insurers, for the most part, suggested dentists handle the increase in cost.

The survey, in turn, reported that 14 percent of the practices surveyed terminated their participation in capitation plans because of the increased costs associated with OSHA regulations and infection control.

Some17 percent of the practices surveyed terminated relations with Medicaid programs.

The costs are real.

Having shared the survey findings with the insurance industry and plan purchasers, the ADA last month fulfilled its 1992 House of Delegates directive.

That directive included sharing the survey with government policy makers as well.

Dr. Al Guay, associate executive director, ADA Division of Dental Practice; Kathleen Todd, ADA associate general counsel; and Carole Kolstad, director, ADA Federal Agency Relations, recently met with OSHA representatives to discuss the result of the survey.

Reported Dr. Guay, "We assured them that we weren't there to rehash the bloodborne pathogen standard, but that there are a number of new issues coming down the pike.

Citing the survey as a model, the ADA offered its assistance in determining the cost of dental office compliance for any future regulations.

Added Dr. Gaines, "There's no shortage of issues currently being looked at by OSHA involving regulations with regard to tuberculosis, ergonomics and indoor air quality."

"All the OSHA concerns I've mentioned are perfectly legitimate safety concerns, of course," he continued, "but that

doesn't necessarily mean the proposals that stem from them will be legitimate."

"In that regard, I want to take advantage of this opportunity to make a personal request [of the insurance industry]. The next time we see a proposal that drives up costs while providing no real benefits to the public, let's work together and let the people who make the decisions know where we stand."

[Add. 147]

American Association of Orthodontists

April 23, 1991

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Civil Rights Division
Coordination and Review Section
P.O. Box 06118
Washington, D.C. 20036-6118
4/24/91

John L. Wodatch
Office on the Americans
with Disabilities Act
Civil Rights Division
U. S. Department of Justice
Rulemaking Docket 003
P.O. Box 75087
Washington, D.C. 20213

Re: **Comments of the American Association of Orthodontists on the Proposed Rule on Nondiscrimination on the Basis of Disability by Public Accommodations and in Commercial Facilities; 28 CFR Part 36, 56 Fed. Reg. 7451 (February 22, 1991)**

Dear Mr: Wodatch:

The American Association of Orthodontists ("Association" or "AAO") is sensitive to the needs of the disabled and supportive of the intent of the Americans with Disabilities Act to make available to disabled Americans a quality, independent lifestyle. The Council on Governmental Affairs and the Board of Trustees of the AAO has reviewed the proposed rule and hereby submit its written comments on the regulations proposed by the Department of Justice implementing Title III of the Act.

The AAO is the recognized specialty organization formed more than eighty years ago to serve the orthodontic needs of the American public. With over 11,000 members, the Association represents over 90 percent of the orthodontists in this country. Orthodontics is the branch of dentistry that specializes in the diagnosis, prevention, and treatment of dental and facial irregularities. The practice of orthodontics requires corrective appliance (braces) to bring teeth, lips and jaws into proper alignment and achieve facial balance. Orthodontists limit their practice to orthodontic treatment. In addition to their dental degree, orthodontists complete a three or two year advanced education program in orthodontics sponsored by a program accredited by the Commission on Dental Accreditation.

[Add. 148]

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[page 5] practices, or procedures will mitigate the risk."

If contagious diseases are included under the definition of disability, then HIV disease should not be included as it presents a direct threat. There are numerous contagious diseases and the DOJ has included only HIV disease in the listing of contagious diseases. For some contagious diseases, because of the number of years the disease has been studied by the medical community, there is a vast amount of medical knowledge accumulated on the disease and a consensus by the medical community and the Centers for Disease Control on appropriate precautions, including barrier techniques, to prevent the spread of the disease. At this time, since HIV disease is a recently identified contagious disease, much uncertainty surrounds what precautions, including barrier techniques, if any, can prevent the transmission of HIV disease. This is of particular concern to health care providers.

There has been one documented case of transmission of HIV disease to a patient from a HIV positive dentist. This well-publicized Florida case is currently being considered by the

Centers for Disease Control. The American Dental Association has issued a policy statement calling for HIV infected dentists to refrain from performing invasive procedures or to disclose their seropositive status. Some contagious diseases may not pose a direct threat. However, at this time scientific evidence indicates that HIV disease poses a direct threat and significant risk.

The Association recommends utilization of appropriate barrier and sterilization techniques, as recommended by the Centers for Disease Control and required by the Occupational Safety and Health Administration to protect patients, orthodontic auxiliaries and orthodontists. The Association asserts that all patients should be required to disclose, as a part of the patient history record, their health history relating to contagious diseases, including HIV disease and hepatitis. Orthodontists must know the health status of their patients in order to ensure proper care and to know if additional barrier precautions are indicated. The DOJ is urged to amend its rule to specifically recognize that this type of health history disclosure is not discrimination.

The Association supports the language contained under the definition of "direct threat" and encourages the deletion of any reference to contagious or noncontagious disease under the definition of "disability." The Association asserts that HIV disease poses a "direct threat" to the health and safety of others, and it is inappropriate to list it under the definition of "disability."

[Add. 149]

[page 6] Numerous public accommodations will interpret the "direct threat" provision to authorize exclusion of individuals with contagious or noncontagious diseases. If the rule includes a reference to contagious diseases then litigation will result or the public will be exposed to the spread of contagious and sometimes fatal diseases.

In addition to the health care provider setting, contagious diseases pose risks to other public accommodations, for example day care centers and schools. Currently most day care centers and schools have policies that prohibit children with contagious disease from attending the center or school. In a situation where the facility provides care for infants and toddlers, who routinely put all objects in their mouths, it would be virtously (sic) impossible to protect the other children from exposures to contagious diseases.

There is a need to balance the rights of non-contagious Americans, including health care providers and patients, who may be exposed to risks of disease with those Americans who have diseases.

It should be noted that the broad definition of "disability" proposed by the DOJ will serve to pre-empt numerous state laws and regulations. Not only have some state civil rights agencies excluded contagious diseases from the definition of "disability" or "handicap," but there are also other state laws that prohibit health care providers from exposing patients to contagious diseases.

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[Add. 150]

Table 11. Health care workers with documented and possible occupationally acquired AIDS/HIV infection, by occupation, reported through June 1995, United States[1]

Occupation	Documented occupational transmission[2] No.	Possible occupational transmission[3] No.
Dental worker, including dentist	-	6
Embalmer/morgue technician	-	2
Emergency medical technician/paramedic	-	9
Health aide/attendant	1	11
Housekeeper/maintenance worker	1	7
Laboratory technician, clinical	15	14
Laboratory technician, nonclinical	3	0
Nurse	16	23
Physician, nonsurgical	6	10
Physician, surgical	-	4
Respiratory therapist	1	2
Technician, dialysis	1	2
Technician, surgical	2	1
Technician/therapist, other than those listed above	-	4
Other health care occupations	-	2
Total	**46**	**97**

[1]Health care workers are defined as those persons, including students and trainees, who have worked in a health care, clinical, or HIV laboratory setting at any time since 1978. See *MMWR* 1992;41:823-25.

[2]Health care workers who had documented HIV seroconversion after occupational exposure or had other laboratory evidence of occupational infection; 40 had percutaneous exposure, 4 had mucocutaneous exposure, 1 had both percutaneous and mucocutaneous exposures, and 1 had an unknown route of exposure. Forty-one exposures were to blood from an HIV-infected person, 1 to visibly bloody fluid, 1 to an unspecified fluid, and 3 to concentrated virus in a laboratory. Twenty of these health care workers developed AIDS.

[3]These health care workers have been investigated and are without identifiable behavioral or transfusion risks; each reported percutaneous or mucocutaneous occupational exposures to blood or body fluids, or laboratory solutions containing HIV, but HIV seroconversion specifically resulting from an occupational exposure was not documented.

[Add. 151]

JAMA
The Journal of the American Medical Association

Editor-In Chief
George D. Lundberg, MD

Original Contribution - July 10, 1996

Prevalence of HIV Infected in the United States, 1984 to 1992

[References and Tables omitted]

John M. Karon, PhD; Philip S. Rosenberg, PhD; Geraldine McQuillan, PhD; Meena Khare, MS; Marta Gwinn, MD, MPH; Lyle R. Petersen, MD, MPH

Objective.—To estimate the number of persons infected with the human immunodeficiency virus (HIV) living in the United States and the change in HIV infection prevalence since 1984.

Design.—We estimated HIV prevalence from 3 data sources. We estimated past HIV infection rates from a statistical procedure based on national acquired immunodeficiency syndrome (AIDS) case surveillance data and estimates of the time from HIV infection to AIDS diagnosis. We also analyzed HIV prevalence data from 2 national surveys, a survey of childbearing women and a household survey of current health status. We used other data sources to adjust these survey estimates to include groups not covered in the surveys.

Results.—Approximately 0.3% of US residents (650,000-900,000 persons) were infected with HIV in 1992. Approximately 0.6% of men (including adolescent boys 13 years of age) were infected, including approximately 2% of non-Hispanic black men and 1% of Hispanic men. Approximately 0.1% of women (including adolescent girls 13 years of age) were infected, including approximately 0.6% of non-Hispanic

black women. Approximately half of all infected persons were men who had sex with men, and one fourth were injecting drug users. The prevalence of HIV infection increased from 1984 to 1992, with a greater relative increase among women than men.

Conclusions.—The 3 different data sources and methods are consistent in estimating that 650,000 to 900,000 persons were infected with HIV in the United States in 1992. Among adolescents and adults of both sexes, the proportion infected was substantially higher among non-Hispanic blacks and Hispanics than among non-Hispanic whites. HIV-related illness will be a major clinical and public health problem in the United States for years to come.

JAMA. 1996;276:126-131

hhttp://www.ama-assn.org/sci-pubs/journals/archive/jama/vol_276/no_2/oc5a84.htm

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[Add. 152]

ESTIMATES of the number and characteristics of persons infected with the human immunodeficiency virus (HIV) in the United States provide a basis for monitoring the course of the HIV epidemic, for focusing prevention efforts on those at greatest risk for HIV infection, and for predicting future HIV-related health care and other service needs. Most estimates of the size of the HIV epidemic have used back-calculation or extrapolation from national surveys of HIV prevalence in specific population groups. Back-calculation is a statistical procedure that estimates the number and distribution over time of HIV infections required to account for the observed number of diagnosed cases of the acquired immunodeficiency syndrome (AIDS). Using the limited seroprevalence data available in 1986

and rough estimates of the sizes of the populations at risk for infection, the US Public Health Service estimated that 1.0 million to 1.5 million US residents were infected with HIV. In 1990, updated US Public Health Service estimates based on both back-calculation and surveys suggested that 800,000 to 1.2 million US residents were infected. Results from back-calculation also suggested that the prevalence in 1986 had actually been approximately 750,000. Since 1990, more data have become available, including additional data from HIV seroprevalence surveys and AIDS case surveillance data. Back-calculation methods have also been improved.

In this report, we present new estimates of HIV prevalence in the US population, including estimates by sex, race/ethnicity, geographic region, and behavioral risk. We also present estimates of prevalence trends since 1984 by sex and by behavioral risk. Our estimates are the first synthesis of numerical estimates of HIV prevalence from multiple estimation procedures. The estimates based on back-calculation include the first estimates by risk for HIV infection. The estimates of HIV prevalence among women include the first estimates based on the Survey in Childbearing Women (SCBW), the only national population-based HIV seroprevalence survey. We also provide the first estimates of HIV prevalence by geographic region and of prevalence among persons not living in households.

METHODS

We estimated total HIV prevalence and prevalence by sex for the US population by adding estimates for children (<13 years of age) to estimates for adults (including adolescents ≥ 13 years of age). For children, we used estimates based on data from the SCBW and from AIDS case surveillance. For adolescents and adults, we estimated HIV prevalence using back-calculation, extrapolation from the SCBW, and HIV prevalence estimates from the Third National Health and Nutrition Examination Survey (NHANES III). Our methods are described in detail in a technical report.

In this report, we define prevalence as the number of persons living

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Original Contribution - July 10, 1996. Prevalence of HIV Infection in the United States.... Page 6 of 15

[page 6] and persons not living in households. We estimated the HIV prevalence among persons aged 13 years or older by dividing the previous range by the proportion of US AIDS cases (excluding children and residents of Puerto Rico and US territories) diagnosed from 1991 through 1994 among persons aged 18 to 59 years. We added an estimate of HIV prevalence in Puerto Rico and US territories based on SCBW data. We also added an estimate of HIV prevalence among persons not living in households (prisoners, hospital patients, and homeless persons) based on data from a variety of sources.

Participation rates in NHANES III were 64% to 72% among groups defined by race/ethnicitiy and sex. It is likely that the HIV prevalence rate was higher among those who did not participate than among those who did. We conducted a sensitivity analysis to estimate the effect of a higher HIV prevalence rate among nonparticipants. We assumed that the HIV prevalence rate among men who did not participate was 1.5 to 2.5 times that among men who did participate. This assumption is consistent with data on HIV behavioral risk among a random sample of nonparticipants in a pilot study for a national household HIV seroprevalence survey.

RESULTS

Results from back-calculation and from the SCBW are consistent with an estimate of 650,000 to 900,000 HIV infected persons living in the United States in 1992, of whom 525,000 to 750,000 were men and 120,000 to 160,000 were women. These ranges, which include all living persons with AIDS already diagnosed, correspond to HIV prevalence rates of 2.5 to 3.5 per 1000 for all US residents, 4.2 to 6.0 per 1000 for males, and 0.9 to 1.2 per 1000 for females. The estimate based on each method includes 10,000 infected children, half boys and half girls. Use of more recent data has almost no effect on the estimates based on the SCBW. Both the lower and upper bounds for the number of infected women change by less than 2000 persons if we use the period 1992 through 1994 instead of 1991 through 1993.

The plausible range for HIV prevalence based on NHANES III is wider than that based on back-calculation or on the SCBW because of the relatively small number of seropositive persons among the participants in NHANES III. The estimates based on NHANES III included an estimated 45,000 to 80,000 and 7000 to 15,000 living HIV-infected adult and adolescent men and women, respectively, who did not live in households. The estimates in Table 1 based on NHANES III do not include adjustments for possible higher HIV prevalence among nonparticipants. The sensitivity analysis (see "Design") suggests that a higher prevalence rate among men who did not participate might have caused NHANES III to underestimate HIV prevalence by 60,000 to 190,000 persons. In addition, some HIV-infected persons with diagnosed AIDS-OIs may have declined to participate in NHANES III for personal health reasons. Because there were more than 100,000

HIV-infected persons with diagnosed AIDS-OIs in the United States in January 1993 (Timothy A. Green, PhD, CDC, unpublished data, 1996), this is also a potential source of substantial bias. Because it is likely that the estimates based on NHANES III are too small, we based our estimate of the plausible range for HIV prevalence among US residents on the ranges from back-calculation and from the SCBW. Nevertheless, after adjustment for potential biases (such as persons who refused to participate for reasons related and unrelated to health), the estimated ranges from NHANES III would be likely to include our estimate of 650,000 to 900,000 HIV-infected US residents derived from other analyses using back-calculation and the SCBW.

The HIV prevalence rates estimated from back-calculation from back-calculation vary by sex and race/ethnicity among adults and adolescents. Approximately 2% of non-Hispanic black men, 1% of Hispanic men, and 0.6% of non-Hispanic black women are infected. Among non-Hispanic whites, the HIV prevalence rate estimated from back-calculation is approximately 10 times higher among men than among women. Among non-Hispanic blacks and Hispanics, the corresponding sex ratio is approximately 4:1.

The HIV prevalence rates among adults and adolescents estimated from the SCBW are lowest in the Midwest and highest in the Northeast and in Puerto Rico. The male-to-female prevalence rate ratio is approximately 10:1 in the West but less than 5:1 in the Northeast and South.

The back-calculation analyses suggest that approximately half of all HIV-infected persons in 1992 were men who have sex with men and who were not also IDUs. By 1992, approximately 25%

of infected persons were IDUs who were not also men who have sex with men, and approximately 15% became infected through heterosexual contact. The prevalence of HIV infection increased from the end of 1986 to the end of 1992 in each risk group shown, but the relative increase among men who have sex with men was less than that among other groups. The back-calculation results indicate that prevalence increased most rapidly from the end of 1986 to the end of 1992 among persons infected through heterosexual contact, but these estimates probably overstate both the HIV prevalence (especially for 1992) and the relative increase in HIV prevalence among these persons as a result of some misclassification of mode of transmission, as discussed below.

HIV prevalence increased among women and adolescent girls from the end of 1984 to the end of 1992. Prevalence also increased among men and adolescent boys from the end of 1984 to the end of 1986 and may have increased from the end of 1986 to the end of 1992, but the relative increases were smaller than those among women.

COMMENT

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[Add. 155]

Our results suggest that 650,000 to 900,000 US residents were living with HIV infection in 1992. National serosurveillance studies generally show stable HIV prevalence rates from 1990 through 1993. These stable rates suggest that, although HIV prevalence increased from 1986 to 1992 in many groups defined by demographic characteristics or risk, most of the increase occurred by 1990 in many of these groups. These stable rates

also suggest that the results reported herein are likely to describe HIV prevalence in the United States at the end of 1993. Our national prevalence estimates are consistent with another recent estimate that 700,000 US residents are currently infected with HIV.

Our estimated range of persons living with HIV infection is lower than the previous US Public Health Service estimate of 800,000 to 1,200,000 infected persons as of 1989, although the ranges from these 2 estimates overlap. In addition to the stable rates observed in the seroprevalence studies, there are other reasons to believe that these differences reflect differences in the data and methods used to obtain these estimates, rather than a reduction in the number of HIV-infected persons in the United States.

The results based on back-calculation in this report are likely to be more accurate than those made in 1989. Temporal trends in AIDS incidence in the United States vary among birth cohorts. The back-calculation results in this report use this information, as well as the dependence of the incubation period on age at time of infection, whereas the approaches used before did not. In addition, these estimates are based on 3 more years of AIDS incidence data. In the United States, the rate of increase in incidence based on the 1987 surveillance definition was less from 1989 through 1992 than from 1981 through 1988 among men who have sex with men, among men who have sex with men and who are also IDUs, and among other IDUs (Timothy A. Green, PhD, CDC, unpublished data, 1996). The decline in the estimated HIV prevalence derived from back-calculation reflects this decline in the rate of AIDS diagnoses.

Differences between the HIV prevalence estimates derived from back-calculation analyses done in 1989 and the current analyses also result from changes in the assumptions about the proportion of HIV-infected persons whose AIDS diagnoses eventually are reported and about the incubation distribution. The previous estimates assumed that AIDS is eventually reported for 75% of

all HIV-infected persons, approximately 10 percentage points less than the 80% to 85% used in this analysis. This change reduces the estimate of HIV seroprevalence by approximately 10%. The incubation distributions used for the current analysis are similar to those used for most of the analyses done in 1989, except that the current analysis includes effects of prophylactic therapy. This change would tend to lengthen the estimated time from HIV infection to AIDS-OI diagnosis and hence increase our estimate of

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[Add. 156]

Report on the Global HIV/AIDS Epidemic

DECEMBER 1997

[Drawing omitted]

EMBARGOED
until
26 November, 1997, 10:00 G.M.T.

Global
HIV/AIDS and STD
Surveillance

Surveillance of HIV/AIDS and STDs is conducted as a joint effort of UNAIDS and WHO. The data in this report have been compiled by the UNAIDS/WHO Working Group on Global HIV/AIDS and STD Surveillance in collaboration with National AIDS Programmes.

[Add. 157]

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Over 27 million do not know they are infected

UNAIDS estimates that of the some 30 million people currently living with HIV, the vast majority have no idea they are infected. As with so many other features of the epidemic, when it comes to knowledge of HIV status there is a gaping divide between the developing and industrialized world.

In the United States, the Centers for Disease Control and Prevention (CDC) estimate that two-thirds of people living with HIV know about their infection. In Germany, the proportion of AIDS patients who knew their HIV status at least six months before an AIDS diagnosis remained steady at close to three-quarters in 1994 and 1995. Since 1995, when drugs that can delay the onset of AIDS came onto the market, the proportion has actually dropped to two-thirds. This is because people who knew their status early have already been treated and have been able to delay the onset of AIDS.

[Add. 158]

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pathogen or that it can be transmitted by aerosols in the dental office. App. 256 (p.55). Dr. Kuvin testified that "HIV is a disease of low infectivity," and that the transfer of blood or bodily fluids from an HIV-positive person usually does not transmit HIV to the recipient. App. 257 (pp. 59, 64).[4] Dr. Kuvin explicitly rejects Dr. Bragdon's mischaracterization of HIV as "highly infectious." App. 267 (p. 203).

According to the United States Centers for Disease Control and Prevention (CDC), which monitors occupational transmission of HIV in the health care setting (including among dental health care workers):

> Despite the fact that probably over one billion dental procedures have been performed by over 250,000 dental health care workers in the past fourteen years, and despite the fact that approximately 1,000,000 individuals in the United States are estimated to be infected with HIV, there has never been a documented case of HIV transmission from infected patient to dental health care worker, nor from infected patient to non-infected patient.

App. 105, 112, para. 16. According to the CDC, the future probability of HIV transmission from patient to dental health

care worker is too low even to be quantifiable. App. 394, para. 3.

6. The Standard Infection Control Procedures Are Designed to Prevent Transmission of Bloodborne Viruses in <u>All</u> Settings.

The majority of patients living with HIV and other

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[4]Not all bloodborne pathogens are alike. In contrast to HIV, Dr. Kuvin testified that Hepatitis B Virus (HBV), another virus transmitted through blood and bodily fluids, is "highly infectious, 100 more times infectious than HIV, easily spread and much more easily spread than HIV." App. 260 (p. 103). According to Dr. Kuvin, saliva or sweat cannot transmit HIV, but can transmit HBV. App. 256 (p. 58).

Recommendations for Prevention of HIV Transmission in Health-Care Settings (Morbidity and Mortality Weekly Report 1987; 36 [no 2S].)

Introduction

Human immunodeficiency virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS), is transmitted through sexual contact and exposure to infected blood or blood components and perinatally from mother to neonate. HIV has been isolated from blood, semen, vaginal secretions, saliva, tears, breast milk, cerebrospinal fluid, amniotic fluid, and urine and is likely to be isolated from other body fluids, secretions, and excretions. However, epidemiologic evidence has implicated only blood, semen, vaginal secretions, and possibly breast milk in transmission.

The increasing prevalence of HIV increases the risk that health-care workers will be exposed to blood from patients infected with HIV, especially when blood and body-fluid precautions are not followed for all patients. Thus, this document emphasizes the need for health-care workers to consider all patients as potentially infected with HIV and/or other blood-borne pathogens and to adhere rigorously to infection-control precautions for minimizing the risk of exposure to blood and body fluids of all patients.

The recommendations contained in this document consolidate and update CDC recommendations published earlier for preventing HIV transmission in health-care settings: precautions for clinical and laboratory staffs (1) and precautions for health-care workers and allied professionals (2); recommendations for preventing HIV transmission in the workplace (3) and during invasive procedures (4); recommendations for preventing possible transmission of HIV from tears (5); and recommendations for providing dialysis treatment for HIV-infected patients (6). These recommendations also update

portions of the "Guideline for Isolation Precautions in Hospitals" (7) and reemphasize some of the recommendations contained in "Infection Control Practices for Dentistry" (8). The recommendations contained in this document have been developed for use in health-care settings and emphasize the need to treat blood and other body fluids from all patients as potentially infective. These same prudent precautions also should be taken in other settings in which persons may be exposed to blood or other body fluids.

Definition of Health-Care Workers

Health-care workers are defined as persons, including students and trainees, whose activities involve contact with patients or with blood or other body fluids from patients in a health-care setting.

Health-Care Workers with AIDS

As of July 10, 1987, a total of 1,875 (5.8%) of 32,395 adults with AIDS, who had been reported to the CDC national surveillance system and for whom occupational information was available, reported being employed in a health-care or clinical laboratory setting. In comparison, 6.8 million persons - representing 5.6% of the U.S. labor force - were employed in health services. Of the health-care workers with AIDS, 95% have been reported to exhibit high-risk behavior; for the remaining 5%, the means of HIV acquisition was undetermined. Health-care workers with AIDS were significantly more likely than other workers to have an undetermined risk (5% versus 3%, respectively). For both health-care and non-health-care workers with AIDS, the proportion with an undetermined risk has not increased since 1982.

AIDS patients initially reported as not belonging to recognized risk groups are investigated by state and local health departments to determine whether possible risk factors exist. Of all health-care workers with AIDS reported to CDC who were initially

characterized as not having an identified risk and for whom follow-up information was available, 66% have been reclassified because risk factors were identified or because the patient was found not to meet the surveillance case definition for AIDS. Of the 87 health-care workers currently categorized as having no identifiable risk, information is incomplete on 16 (18%) because of death or refusal to be interviewed; 38 (44%) are still being investigated. The remaining 33 (38%) health-care workers were interviewed or had other follow-up information available. The occupations of these 33 were as follows: five physicians (15%), three of whom were surgeons; one dentist (3%); three nurses (9%); nine nursing assistants (27%); seven housekeeping or maintenance workers (21%); three clinical laboratory technicians (9%); one therapist (3%); and four others who did not have contact with patients (12%). Although 15 of these 33 health-care workers reported parenteral and/or other non-needlestick exposure to blood or body fluids from patients in the 10 years preceding their diagnosis of AIDS, none of these exposures involved a patient with AIDS or know HIV infection.

Risk to Health-Care Workers of Acquiring HIV in Health-Care Settings

Health-care workers with documented percutaneous or mucous-membrane exposures to blood or body fluids of HIV-infected patients have been prospectively evaluated to determine the risk of infection after such exposures. As of June 30, 1987, 883 health-care workers have been tested for antibody to HIV in an ongoing surveillance project conducted by CDC (9). Of these, 708 (80%) had percutaneous exposures to blood, and 175 (20%) had a mucous membrane or an open would contaminated by blood or body fluid. Of 396 health-

care workers, each of whom had only a convalescent-phase serum sample obtained and tested ≥90 days postexposure, one - for whom heterosexual transmission could not be ruled out - was

seropositive for HIV antibody. For 425 additional health-care workers, both acute - and convalescent-phase serum samples were obtained and tested; none of 74 health-care workers with nonpercutaneous exposures seroconverted, and three (0.9%) of 351 with percutaneous exposures seroconverted. None of these health-care workers had other documented risk factors for infection.

Two other prospective studies to assess the risk of nosocomial acquisition of HIV infection for health-care workers are ongoing in the United States. As of April 30, 1987, 332 health-care workers with a total of 453 needlestick or mucous-membrane exposures to the blood or other body fluids of HIV-infected patients were tested for HIV antibody at the National Institutes of Health (10). These exposed workers included 103 with needlestick injuries and 229 with mucous-membrane exposures; none had seroconverted. A similar study at the University of California of 129 health-care workers with documented needlestick injuries or mucous-membrane exposures to blood or other body fluids from patients with HIV infection has not identified any seroconversions (11). Results of a prospective study in the United Kingdom identified no evidence of transmission among 150 health-care workers with parenteral or mucous-membrane exposures to blood or other body fluids, secretions, or excretions from patients with HIV infection (12).

In addition to health-care workers enrolled in prospective studies, eight persons who provided care to infected patients and denied other risk factors have been reported to have acquired HIV infection. Three of these health-care workers had needlestick exposures to blood from infected patients (13-15). Two were persons who provided nursing care to infected persons; although neither sustained a needlestick, both had extensive contact with blood or other body fluids, and neither observed recommended barrier precautions (16,17). The other three were health-care workers with non-needlestick exposures to blood from infected patients (18). Although the exact route of transmission for these last three infections is not known, all three

persons had direct contact of their skin with blood from infected patients, all had skin lesions that may have been contaminated by blood, and one also had a mucous-membrane exposure.

A total of 1,231 dentists and hygienists, many of whom practiced in areas with many AIDS cases, participated in a study to determine the prevalence of antibody to HIV; one dentist (0.1%) had HIV antibody. Although no exposure to a known HIV-infected person could be documented, epidemiologic investigation did not identify any other risk factor for infection. The infected dentist, who also had a history of sustaining needlestick injuries and trauma to his hands, did not routinely wear gloves when providing dental care (19).

Precautions to Prevent Transmission of HIV

Universal Precautions

Since medical history and examination cannot reliably identify all patients infected with HIV or other blood-borne pathogens, blood and body-fluid precautions should be consistently used for **all** patients. This approach, previously recommended by CDC (3,4), and referred to as "universal blood and body-fluid precautions" or "universal precautions," should be used in the care of **all** patients, especially including those in emergency-care settings in which the risk of blood exposure is increased and the infection status of the patient is usually unknown (20).

1. All health-care workers should routinely use appropriate barrier precautions to prevent skin and mucous-membrane exposure when contact with blood or other body fluids of any patient is anticipated. Gloves should be worn for touching blood and body fluids, mucous membranes, or non-intact skin of all patients, for handling items or surfaces soiled with blood or body fluids, and for performing venipuncture and other vascular access procedures. Gloves should be changed after contact with each patient. Masks and protective eyewear or face shields should be worn during procedures that are

likely to generate droplets of blood or other body fluids to prevent exposure of mucous membranes of the mouth, nose, and eyes. Gowns or aprons should be worn during procedures that are likely to generate splashes of blood or other body fluids.

2. Hands and other skin surfaces should be washed immediately and thoroughly if contaminated with blood or other body fluids. Hands should be washed immediately after gloves are removed.

3. All health-care workers should take precautions to prevent injuries caused by needles, scalpels, and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles; and when handling sharp instruments after procedures. To prevent needlestick injuries, needles should not be recapped, purposely bent or broken by hand, removed from disposable syringes, or otherwise manipulated by hand. After they are used, disposable syringes and needles, scalpel blades, and other sharp items should be placed in puncture-resistant containers for disposal; the puncture-resistant containers should be located as close as practical to the use area. Large-bore reusable needles should be placed in a puncture-resistant container for transport to the reprocessing area.

4. Although saliva has not been implicated in HIV transmission, to minimize the need for emergency mouth-to-mouth resuscitation, mouthpieces, resuscitation bags, or other ventilation devices should be available for use in areas in which the need for resuscitation is predictable.

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5. Health-care workers who have exudative lesions or weeping dermatitis should refrain from all direct patient care and from handling patient-care equipment until the condition resolves.

6. Pregnant health-care workers are not known to be at greater risk of contracting HIV infection than health-care workers who are not pregnant; however, if a health-care worker develops HIV infection during pregnancy, the infant is at risk of infection resulting from perinatal transmission. Because of this risk, pregnant health-care workers should be especially familiar with and strictly adhere to precautions to minimize the risk of HIV transmission.

Implementation of universal blood and body-fluid precautions for **all** patients eliminates the need for use of the isolation category of "Blood and Body Fluid Precautions" previously recommended by CDC (7) for patients known or suspected to be infected with blood-borne pathogens. Isolation precautions (e.g., enteric, "AFB" [7]) should be used as necessary if associated conditions, such as infectious diarrhea tuberculosis, are diagnosed or suspected.

Precautions for Invasive Procedures

In this document, an invasive procedure is defined as surgical entry into tissues, cavities, or organs or repair of major traumatic injuries 1) in an operating or delivery room, emergency department, or outpatient setting, including both physicians' and dentists' offices; 2) cardiac catheterization and angiographic procedures; 3) a vaginal or cesarean delivery or other invasive obstetric procedure during which bleeding may occur, or 4) the manipulation, cutting, or removal of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists. The universal blood and body-fluid precautions listed above, combined with the precautions listed below, should be the minimum precautions for **all** such invasive procedures.

1. All health-care workers who participate in invasive procedures must routinely use appropriate barrier precautions to prevent skin and mucous-membrane contact with blood and other body fluids of all patients. Gloves and surgical

masks must be worn for all invasive procedures. Protective eyewear or face shields should be worn for procedures that commonly result in the generation of droplets, splashing of blood or other body fluids, or the generation of bone chips. Gowns or aprons made of materials that provide an effective barrier should be worn during invasive procedures that are likely to result in the splashing of blood or other body fluids. All health-care workers who perform or assist in vaginal or cesarean deliveries should wear gloves and gowns when handling the placenta or the infant until blood and amniotic fluid have been removed from the infant's skin and should wear gloves during post-delivery care of the umbilical cord.

2. If a glove is torn or a needlestick or other injury occurs, the glove should be removed and a new glove used as promptly as patient safety permits; the needle or instrument involved in the incident should also be removed from the sterile field.

Precautions for Dentistry*

Blood, saliva, and gingival fluid from **all** dental patients should be considered infective. Special emphasis should be placed on the following precautions for preventing transmission of blood-borne pathogens in dental practice in both institutional and non-institutional settings.

1. In addition to wearing gloves for contact with oral mucous membranes of all patients, all dental workers should wear surgical masks and protective eyewear or chin-length plastic face shields during dental procedures in which splashing or spattering of blood, saliva, or gingival fluids is likely. Rubber dams, high-speed evacuation, and proper patient positioning, when appropriate, should be utilized to minimize generation of droplets and spatter.

2. Handpieces should be sterilized after use with each patient, since blood, saliva, or gingival fluid of patients may be aspirated into the handpiece or waterline. Handpieces that cannot be

sterilized should at least be flushed, the outside surface cleaned and wiped with a suitable chemical germicide, and then rinsed. Handpieces should be flushed at the beginning of the day and after use with each patient. Manufacturers' recommendations should be followed for use and maintenance of waterlines and check valves and for flushing of handpieces. The same precautions should be used for ultrasonic scalers and air/water syringes.

3. Blood and saliva should be thoroughly and carefully cleaned from material that has been used in the mouth (e.g., impression materials, bite registration), especially before polishing and grinding intra-oral devices. Contaminated materials, impressions, and intra-oral devices should also be cleaned and disinfected before being handled in the dental laboratory and before they are placed in the patient's mouth. Because of the increasing variety of dental materials used intra-orally, dental workers should consult with manufacturers as to the stability of specific materials when using disinfection procedures.
4. Dental equipment and surfaces that are difficult to disinfect (e.g., light handles or X-ray-unit heads) and that may become contaminated should be wrapped with impervious-backed paper, aluminum foil, or clear plastic wrap. The coverings should be removed and discarded, and clean coverings should be put in place after use with each patient.

Precautions for Autopsies or Morticians' Services

In addition to the universal blood and body-fluid precautions listed above, the following precautions should be used by persons performing postmortem procedures:

1. All persons performing or assisting in postmortem

*General infection-control precautions are more specifically addressed in previous recommendations for infection-control practices for dentistry (8).

procedures should wear gloves, masks, protective eyewear, gowns, and waterproof aprons.

2. Instruments and surfaces contaminated during postmortem procedures should be decontaminated with an appropriate chemical germicide.

Precautions for Dialysis

Patients with end-stage renal disease who are undergoing maintenance dialysis and who have HIV infection can be dialyzed in hospital-based or free-standing dialysis units using conventional infection-control precautions (*21*). Universal blood and body-fluid precautions should be used when dialyzing **all** patients.

Strategies for disinfecting the dialysis fluid pathways of the hemodialysis machine are targeted to control bacterial contamination and generally consist of using 500-750 parts per million (ppm) of sodium hypochlorite (household bleach) for 30-40 minutes or 1.5%-2.0% formaldehyde overnight. In addition, several chemical germicides formulated to disinfect dialysis machines are commercially available. None of these protocols or procedures need to be changed for dialyzing patients infected with HIV.

Patients infected with HIV can be dialyzed by either hemodialysis or peritoneal dialysis and do not need to be isolated from other patients. The type of dialysis treatment (i.e., hemodialysis or peritoneal dialysis) should be based on the needs of the patient. The dialyzer may be discarded after each use. Alternatively, centers that reuse dialyzers -- i.e., a specific single-use dialyzer is issued to a specific patient, removed, cleaned, disinfected, and reused several times on the same patient only -- may include HIV-infected patients in the dialyzer-reuse program. An individual dialyzer must never be used on more than one patient.

Precautions for Laboratories[†]

Blood and other body fluids from **all** patients should be considered infective. To supplement the universal blood and body-

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[†]Additional precautions for research and industrial laboratories are addressed elsewhere (*22,23*).

fluid precautions listed above, the following precautions are recommended for health-care workers in clinical laboratories.

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1. All specimens of blood and body fluids should be put in a well-constructed container with a secure lid to prevent leaking during transport. Care should be taken when collecting each specimen to avoid contaminating the outside of the container and of the laboratory form accompanying the specimen.
2. All persons processing blood and body-fluid specimens (e.g., removing tops from vacuum tubes) should wear gloves. Masks and protective eyewear should be worn if mucous-membrane contact with blood or body fluids is anticipated. Gloves should be changed and hands washed after completion of specimen processing.
3. For routine procedures, such as historologic and pathologic studies or microbiologic culturing, a biological safety cabinet is not necessary. However, biological safety cabinets (Class I or II) should be used whenever procedures are conducted that have a high potential for generating droplets. These include activities such as blending, sonicating, and vigorous mixing.
4. Mechanical pipetting devices should be used for manipulating all liquids in the laboratory. Mouth pipetting must not be done.
5. Use of needles and syringes should be limited to situations in which there is no alternative, and the recommendations for preventing injuries with needles outlined under universal precautions should be followed.
6. Laboratory work surfaces should be decontaminated with an appropriate chemical germicide after a spill of blood or other body fluids and when work activities are completed.
7. Contaminated materials used in laboratory tests should be decontaminated before reprocessing or be placed in bags and disposed of in accordance with institutional policies for disposal of infective waste (*24*).
8. Scientific equipment that has been contaminated with blood or other body fluids should be decontaminated and cleaned before being repaired in the laboratory or transported to the manufacturer.

9. All persons should wash their hands after completing laboratory activities and should remove protective clothing before leaving the laboratory.

Implementation of universal blood and body-fluid precautions for **all** patients eliminates the need for warning labels on specimens since blood and other body fluids from all patients should be considered infective.

Environmental Considerations for HIV Transmission

No environmentally mediated mode of HIV transmission has been documented. Nevertheless, the precautions described below should be taken routinely in the care of **all** patients.

Sterilization and Disinfection

Standard sterilization and disinfection procedures for patient-care equipment currently recommended for use (*25,26*) in a variety of health-care settings -- including hospitals, medical and dental clinics and offices, hemodialysis centers, emergency-care facilities, and long-term nursing care facilities -- are adequate to sterilize or disinfect instruments, devices, or other items contaminated with blood or other body fluids from persons infected with blood-borne pathogens including HIV (*21, 23*).

Instruments or devices that enter sterile tissue or the vascular system of any patient or through which blood flows should be sterilized before reuse. Devices or items that contact intact mucous membranes should be sterilized or receive high-level disinfection, a procedure that kills vegetative organisms and viruses but not necessarily large numbers of bacterial spores. Chemical germicides that are registered with the U.S. Environmental Protection Agency (EPA) as "sterilants" may be used either for sterilization or for high-level disinfection depending on contact time.

Medical devices or instruments that require sterilization or disinfection should be thoroughly cleaned before being exposed to the germicide, and the manufacturer's instructions for the use of the germicide should be followed. Further, it is important that the

manufacturer's specifications for compatibility of the medical device with chemical germicides be closely followed. Information on specific label claims of commercial germicides can be obtained by writing to the Disinfectants Branch, Office of Pesticides, Environmental Protection Agency, 401 M Street, SW, Washington, D.C. 20460.

Studies have shown that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than used in practice (*27-30*). Embalming fluids are similar to the types of chemical germicides that have been tested and found to completely inactivate HIV. In addition to commercially available chemical germicides, a solution of sodium hypochlorite (household bleach) prepared daily is an inexpensive and effective germicide. Concentrations ranging from approximately 500 ppm (1:100 dilution of household bleach) sodium hypochlorite to 5,000 ppm (1:10 dilution of household bleach) are effective depending on the amount of organic material (e.g., blood, mucous) present on the surface to be cleaned and disinfected. Commercially available chemical germicides may be more compatible with certain medical devices that might be corroded by repeated exposure to sodium hypochlorite, especially to the 1:10 dilution.

Survival of HIV in the Environment

The most extensive study on the survival of HIV after drying involved greatly concentrated HIV samples, i.e., 10 million tissue-culture infectious doses per milliliter (*31*). This concentration is at least 100,000 times greater than that typically found in the blood or serum of patients with HIV infection. HIV was detectable by tissue-culture techniques 1-3 days after drying, but the rate of inactivation was rapid. Studies performed at CDC have also shown that drying HIV causes a rapid (within several hours) 1-2 log (90%-99%) reduction in HIV concentration. In tissue-culture fluid, cell-free HIV could be detected up to 15 days at room temperature, up to 11 days at 37 C (98.6 F), and up to 1 day if the HIV was cell-associated.

When considered in the context of environmental conditions in health-care facilities, these results do not require any changes in currently recommended sterilization, disinfection, or housekeeping strategies. When medical devices are contaminated

with blood or other body fluids, existing recommendations include the cleaning of these instruments, followed by disinfection or sterilization, depending on the type of medical device. These protocols assume "worst-case" conditions of

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extreme virologic and microbiologic contamination, and whether viruses have been inactivated after drying plays no role in formulating these strategies. Consequently, no changes in published procedures for cleaning, disinfecting, or sterilizing need to be made.

Housekeeping

Environmental surfaces such as walls, floors, and other surfaces are not associated with transmission of infections to patients or health-care workers. Therefore, extraordinary attempts to disinfect or sterilize these environmental surfaces are not necessary. However, cleaning and removal of soil should be done routinely.

Cleaning schedules and methods vary according to the area of the hospital or institution, type of surface to be cleaned, and the amount and type of soil present. Horizontal surfaces (e.g., bedside tables and hard-surfaced flooring) in patient-care areas are usually cleaned on a regular basis, when soiling or spills occur, and when a patient is discharged. Cleaning of walls, blinds, and curtains is recommended only if they are visibly soiled. Disinfectant fogging is an unsatisfactory method of decontaminating air and surfaces and is not recommended.

Disinfectant-detergent formulations registered by EPA can be used for cleaning environmental surfaces, but the actual physical removal of microorganisms by scrubbing is probably at least as important as any antimicrobial effect of the cleaning agent used. Therefore, cost, safety, and acceptability by housekeepers can be the main criteria for selecting any such registered agent. The manufacturer's instructions for appropriate use should be followed.

Cleaning and Decontaminating Spills of Blood or Other Body Fluids

Chemical germicides that are tuberculocidal when used at recommended dilutions can be used to decontaminate spills of blood and other body fluids. Strategies for decontaminating spills of blood and other body fluids in a patient-care setting are different than for spills of cultures or other materials in clinical, public health, or research laboratories. In patient-care areas, visible material should first be removed and then the area should be decontaminated. With large spills of cultured or concentrated infectious agents in the laboratory, the contaminated area should be flooded with a liquid germicide before cleaning, then decontaminated with fresh germicidal chemical. In both settings, gloves should be worn during the cleaning and decontaminating procedures.

Laundry

Although soiled linen has been identified as a source of large numbers of certain pathogenic microorganisms, the risk of actual disease transmission is negligible. Rather than rigid procedures and specifications, hygienic and common-sense storage and processing of clean and soiled linen are recommended (*26*). Soiled linen should be handled as little as possible and with minimum agitation to prevent gross microbial contamination of the air and of persons handling the linen. All soiled linen should be bagged at the location where it was used; it should not be sorted or rinsed in patient-care areas. Linen soiled with blood or body fluids should be placed and transported in bags that prevent leakage. If hot water is used, linen should be washed

with detergent in water at least 71 C (160 F) for 25 minutes. If low-temperature (≤70 C [158 F]) laundry cycles are used, chemicals suitable for low-temperature washing at proper use concentration should be used.

Infective Waste

There is no epidemiologic evidence to suggest that most hospital waste is any more infective than residential waste.

Moreover, there is no epidemiologic evidence that hospital waste has caused disease in the community as a result of improper disposal. Therefore, identifying wastes for which special precautions are indicated is largely a matter of judgment about the relative risk of disease transmission. The most practical approach to the management of infective waste is to identify those wastes with the potential for causing infection during handling and disposal and for which some special precautions appear prudent. Hospital wastes for which special precautions appear prudent include microbiology laboratory waste, pathology waste, and blood specimens or blood products. While any item that has had contact with blood, exudates, or secretions may be potentially infective, it is not usually considered practical or necessary to treat all such waste as infective (*23, 26*). Infective waste, in general, should either be incinerated or should be autoclaved before disposal in a sanitary landfill. Bulk blood, suctioned fluids, excretions, and secretions may be carefully poured down a drain connected to a sanitary sewer. Sanitary sewers may also be used to dispose of other infectious wastes capable of being ground and flushed into the sewer.

Implementation of Recommended Precautions

Employers of health-care workers should ensure that policies exist for:

1. Initial orientation and continuing education and training of all health-care workers -- including students and trainees -- on the epidemiology, modes of transmission, and prevention of HIV and other blood-borne infections and the need for routine use of universal blood and body-fluid precautions for **all** patients.
2. Provision of equipment and supplies necessary to minimize the risk of infection with HIV and other blood-borne pathogens.
3. Monitoring adherence to recommended protective measures. When monitoring reveals a failure to follow recommended precautions, counseling, education, and/or re-training should be provided, and, if necessary, appropriate disciplinary action should be considered.

Professional associations and labor organizations, through continuing education efforts, should emphasize the need for health-care workers to follow recommended precautions.

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Serologic Testing for HIV Infection

Background

A person is identified as infected with HIV when a sequence of tests, starting with repeated enzyme immunoassays (EIA) and including a Western blot of similar, more specific assay, are repeatedly reactive. Persons infected with HIV usually develop antibody against the virus within 6-12 weeks after infection.

The sensitivity of the currently licensed EIA tests is at least 99% when they are performed under optimal laboratory conditions on serum specimens from persons infected for ≥12 weeks. Optimal laboratory conditions include the use of reliable reagents, provision of continuing education of personnel, quality control of procedures, and participation in performance-evaluation programs. Given this performance, the probability of a false-negative test is remote except during the first several weeks after infection, before detectable antibody is present. The proportion of infected persons with a false-negative test attributed to absence of antibody in the early stages of infection is dependent on both the incidence and prevalence of HIV infection in a population (Table 1).

The specificity of the currently licensed EIA tests is approximately 99% when repeatedly reactive tests are considered. Repeat testing of initially reactive specimens by EIA is required to reduce the likelihood of laboratory error. To increase further the specificity of serologic tests, laboratories must use a supplemental test, most often the Western blot, to validate repeatedly reactive EIA results. Under optimal laboratory conditions, the sensitivity of the Western blot test is comparable to or greater than that of a repeatedly reactive EIA, and the Western blot is highly specific when strict criteria are used to interpret the test results. The testing sequence of a repeatedly reactive EIA and a positive Western blot test is highly predictive of HIV infection, even in a population with a low prevalence of infection (Table 2). If the Western blot test result is indeterminant, the testing sequence is considered equivocal for HIV infection.

[Table 1 omitted]

When this occurs, the Western blot test should be repeated on the same serum sample, and, if still indeterminant, the testing sequence should be repeated on a sample collected 3-6 months later. Use of other supplemental tests may aid in interpreting of results on samples that are persistently indeterminant by Western blot.

Testing of Patients

Previous CDC recommendations have emphasized the value of HIV serologic testing of patients for: 1) management of parenteral or mucous-membrane exposures of health-care workers, 2) patient diagnosis and management, and 3) counseling and serologic testing to prevent and control HIV transmission in the community. In addition, more recent recommendations have stated that hospitals, in conjunction with state and local health departments, should periodically determine the prevalence of HIV infection among patients from age groups at highest risk of infection (*32*).

Adherence to universal blood and body-fluid precautions recommended for the care of all patients will minimize the risk of transmission of HIV and other blood-borne pathogens from patients to health-care workers. The utility of routine HIV serologic testing of patients as an adjunct to universal precautions is unknown. Results of such testing may not be available in emergency or outpatient settings. In addition, some recently infected patients will not have detectable antibody to HIV (Table 1).

Personnel in some hospitals have advocated serologic testing of patients in settings in which exposure of health-care workers to large amounts of patients' blood may be anticipated. Specific patients for whom serologic testing has been advocated include those undergoing major operative procedures and those undergoing treatment in critical-care units, especially if they have conditions involving uncontrolled bleeding. Decisions regarding the need to establish testing programs for patients should be made by physicians or individual institutions. In addition, when deemed appropriate, testing of individual patients may be performed on agreement between the patient and the physician providing care.

In addition to the universal precautions recommended for all patients, certain additional precautions for the care of HIV-

infected patients undergoing major surgical operations have been proposed by personnel in some hospitals. For example, surgical procedures on an HIV-infected patient might be altered so that hand-to-hand passing of sharp instruments would be eliminated; stapling instruments rather than

[Table 2 omitted]

hand-suturing equipment might be used to perform tissue approximation; electrocautery devices rather than scalpels might be used as cutting instruments; and, even though uncomfortable, gowns that totally prevent seepage of blood onto the skin of members of the operative team might be worn. While such modifications might further minimize the risk of HIV infection for members of the operative team, some of these techniques could result in prolongation of operative time and could potentially have an adverse effect on the patient.

Testing programs, if developed, should include the following principles:

- Obtaining consent for testing.
- Informing patients of test results, and providing counseling for seropositive patients by properly trained persons.
- Assuring that confidentiality safeguards are in place to limit knowledge of test results to those directly involved in the care of infected patients or as required by law.
- Assuring that identification of infected patients will not result in denial of needed care or provision of suboptimal care.
- Evaluating prospectively 1) the efficacy of the program in reducing the incidence of parenteral, mucous-membrane, or significant cutaneous exposures of health-care workers to the blood or other body fluids of HIV-infected patients and 2) the effect of modified procedures on patients.

Testing of Health-Care Workers

Although transmission of HIV from infected health-care workers to patients has not been reported, transmission during

invasive procedures remains a possibility. Transmission of hepatitis B virus (HBV) -- a blood-borne agent with a considerably greater potential for nosocomial spread -- from health-care workers to patients has been documented. Such transmission has occurred in situations (e.g., oral and gynecologic surgery) in which health-care workers, when tested, had very high concentrations of HBV in their blood (at least 100 million infectious virus particles per milliliter, a concentration much higher than occurs with HIV infection), and the health-care workers sustained a puncture wound while performing invasive procedures or had exudative or weeping lesions or microlacerations that allowed virus to contaminate instruments or open wounds of patients (*33, 34*).

The hepatitis B experience indicates that only those health-care workers who perform certain types of invasive procedures have transmitted HBV to patients. Adherence to recommendations in this document will minimize the risk of transmission of HIV and other blood-borne pathogens from health-care workers to patients during invasive procedures. Since transmission of HIV from infected health-care workers performing invasive procedures to their patients has not been reported and would be expected to occur only very rarely, if at all, the utility of routine testing of such health-care workers to prevent transmission of HIV cannot be assessed. If consideration is given to developing a serologic testing program for health-care workers who perform invasive procedures, the frequency of testing, as well as the issues of consent, confidentially, and consequences of test results -- as previously outlined for testing programs for patients -- must be addressed.

Management of Infected Health-Care Workers

Health-care workers with impaired immune systems resulting from HIV infection or other causes are at increased risk of acquiring or experiencing serious complications of infectious disease. Of particular concern is the risk of severe infection following exposure to patients with infectious diseases that are easily transmitted if appropriate precautions are not taken (e.g., measles, varicella). Any health-care worker with an impaired immune system should be counseled about the potential risk associated with taking care of patients with any transmissible

infection and should continue to follow existing recommendations for infection control to minimize risk of exposure to other infectious agents (*7, 35*). Recommendations of the Immunization Practices Advisory Committee (ACIP) and institutional policies concerning requirements for vaccinating health-care workers with live-virus vaccines (e.g., measles, rubella) should also be considered.

The question of whether workers infected with HIV -- especially those who perform invasive procedures -- can adequately and safely be allowed to perform patient-care duties or whether their work assignments should be changed must be determined on an individual basis. These decisions should be made by the health-care worker's personal physician(s) in conjunction with the medical directors and personnel health service staff of the employing institution or hospital.

Management of Exposures

If a health-care worker has a parenteral (e.g., needlestick or cut) or mucous-membrane (e.g., splash to the eye or mouth) exposure to blood or other body fluids or has a cutaneous exposure involving large amounts of blood or prolonged contact with blood -- especially when the exposed skin is chapped, abraded, or afflicted with dermatitis -- the source patient should be informed of the incident and tested for serologic evidence of HIV infection after consent is obtained. Policies should be developed for testing source patients in situations in which consent cannot be obtained (e.g., an unconscious patient).

If the source patient has AIDS, is positive for HIV antibody, or refuses the test, the health-care worker should be counseled regarding the risk of infection and evaluated clinically and serologically for evidence of HIV infection as soon as possible after the exposure. The health-care worker should be advised to report and seek medical evaluation for any acute febrile illness that occurs within 12 weeks after the exposure. Such an illness -- particularly one characterized by fever, rash, or lymphadenopathy -- may be indicative of recent HIV infection. Seronegative health-care workers should be retested 6 weeks post-exposure and on a periodic basis thereafter (e.g., 12 weeks and 6 months after exposure) to determine whether transmission has occurred. During this follow-up period -- especially the first 6-12 weeks after exposure, when most infected persons are expected to seroconvert -- exposed

health-care workers should follow U.S. Public Health Service (PHS) recommendations for preventing transmission of HIV (*36, 37*).

No further follow-up of a health-care worker exposed to infection as described above is necessary if the source patient is seronegative unless the source patient is at high risk of HIV infection. In the latter case, a subsequent specimen (e.g., 12 weeks following exposure) may be obtained from the health-care worker for antibody

testing. If the source patient cannot be identified, decisions regarding appropriate follow-up should be individualized. Serologic testing should be available to all health-care workers who are concerned that they may have been infected with HIV.

It a patient has a parenteral or mucous-membrane exposure to blood or other body fluid of a health-care worker, the patient should be informed of the incident, and the same procedure outlined above for management of exposures should be followed for both the source health-care worker and the exposed patient.

[References omitted]

Update: Universal Precautions for Prevention of Transmission of Human Immunodeficiency Virus, Hepatitis B Virus, and Other Bloodborne Pathogens in Health-Care Settings (Morbidity and Mortality Weekly Report, 1988; 37:377-382,387,388.)

Introduction

The purpose of this report is to clarify and supplement the CDC publication entitled "Recommendations for Prevention of HIV Transmission in Health-Care Settings (1).*

In 1983, CDC published a document entitled "Guideline for Isolation Precautions in Hospitals" (2) that contained a section entitled "Blood and Body Fluid Precautions." The recommendations in this section called for blood and body fluid precautions when a patient was know or suspected to be infected with bloodborne pathogens. In August 1987, CDC published a document entitled "Recommendations for Prevention of HIV Transmission in Health-Care Settings" (1). In contrast to the 1983 document, the 1987 document recommended that blood and body fluid precautions be consistently used for all patients regardless of their bloodborne infection status. This extension of blood and body fluid precautions to all patients is referred to as "Universal Blood and Body Fluid Precautions" or "Universal Precautions." Under universal precautions, blood and certain body fluids of all patients are considered potentially infectious for human immunodeficiency virus (HIV), hepatitis B virus (HBV), and other bloodborne pathogens.

Universal precautions are intended to prevent parenteral, mucous membrane, and nonintact skin exposures of health-care workers to bloodborne pathogens. In addition, immunization with HBV vaccine is recommended as an important adjunct to universal precautions for health-care workers who have exposures to blood (3,4).

Since the recommendations for universal precautions were published in August 1987, CDC and the Food and Drug Administration (FDA) have received requests for clarification of the following issues: 1) body fluids to which universal precautions apply, 2) use of protective barriers, 3) use of gloves for phlebotomy, 4) selection of gloves for use while observing universal precautions and 5) need for making changes in waste management programs as a result of adopting universal precautions.

Body Fluids to Which Universal Precautions Apply

Universal precautions apply to blood and to other body fluids containing visible blood. Occupational transmission of HIV and HBV to health-care workers by blood is documented (4,5). Blood is the single most important source of HIV, HBV, and other bloodborne pathogens in the occupational setting. Infection control efforts for HIV, HBV, and other bloodborne pathogens must focus on preventing exposures to blood as well as on delivery of HBV immunization.

Universal precautions also apply to semen and vaginal secretions. Although both of these fluids have been implicated in the sexual transmission of HIV and HBV, they have not been implicated in occupational transmission from patient to health-care worker. This observation is not unexpected, since exposure to semen in the usual health-care setting is limited, and the routine practice of wearing gloves for performing vaginal examinations protects health-care workers from exposure to potentially infectious vaginal secretions.

Universal precautions also apply to tissues and to the following fluids: cerebrospinal fluid (CFS), synovial fluid, pleural fluid, peritoneal fluid, pericardial fluid, and amniotic fluid. The risk of transmission of HIV and HBV from these fluids is unknown; epidemiologic studies in the health-care and community setting are currently inadequate to assess the potential risk to health-care workers from occupational exposures to them. However, HIV

has been isolated from CSF, synovial, and amniotic fluid (6-8), and HBsAg has been detected in synovial fluid, amniotic fluid, and peritoneal fluid (9-11). One case of HIV transmission was reported after a percutaneous exposure to bloody pleural fluid obtained by needle aspiration (12). Whereas aseptic procedures used to obtain these fluids for diagnostic or therapeutic purposes protect health-care workers from skin exposures, they cannot prevent penetrating injuries due to contaminated needles or other sharp instruments.

Body Fluids to Which Universal Precautions Do Not Apply

Universal precautions do not apply to feces, nasal secretions, sputum, sweat, tears, urine, and vomitus unless they contain visible blood. The risk of transmission of HIV and HBV from these fluids and materials is extremely low or nonexistent. HIV has been isolated and HBsAg has been demonstrated in some of these fluids; however, epidemiologic studies in the health-care and community setting have not implicated these fluids or materials in the transmission of HIV and HBV infections (13,14). Some of the above fluids and excretions

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*The August 1987 publication should be consulted for general information and specific recommendations not addressed in this update.

represent a potential source for nosocomial and community-acquired infections with other pathogens, and recommendations for preventing the transmission of non-bloodborne pathogens have been published (2).

Precautions for Other Body Fluids in Special Settings

Human breast milk has been implicated in perinatal transmission of HIV, and HBsAg has been found in the milk of mothers infected with HBV (10,13). However, occupational exposure to

human breast milk has not been implicated in the transmission of HIV nor HBV infection to health-care workers. Moreover, the health-care workers will not have the same type of intensive exposure to breast milk as the nursing neonate. Whereas universal precautions do not apply to human breast milk, gloves may be worn by health-care workers in situations where exposures to breast milk might be frequent, for example, in breast milk banking.

Saliva of some persons infected with HBV has been shown to contain HBV-DNA at concentrations 1/1,000 to 1/10,000 of that found in the infected person's serum (15). HbsAg-positive saliva has been shown to be infectious when injected into experimental animals and in human bite exposures (16-18). However, HBsAg-positive saliva has not been shown to be infectious when applied to oral mucous membranes in experimental primate studies (18) or through contamination of musical instruments or cardiopulmonary resuscitation dummies used by HBV carriers (19,20). Epidemiologic studies of nonsexual household contacts of HIV-infected patients, including several small series in which HIV transmission failed to occur after bites or after percutaneous inoculation or contamination of cuts and open wounds with saliva from HIV-infected patients, suggest that the potential for salivary transmission of HIV is remote (5,13,14,21,22). One case report from Germany has suggested the possibility of transmission of HIV in a household setting from an infected child to a sibling through a human bite (23). The bite did not break the skin or result in bleeding. Since the date of seroconversion to HIV was not known for either child in this case, evidence for the role of saliva in the transmission of virus is unclear (23). Another case report suggested the possibility of transmission of HIV from husband to wife by contact with saliva during kissing (24). However, follow-up studies did not confirm HIV infection in the wife (21).

Universal precautions do not apply to saliva. General infection control practices already in existence - including the use of

gloves for digital examination of mucous membranes and endotracheal suctioning, and handwashing after exposure to saliva - should further minimize the minute risk, if any, for salivary transmission of HIV and HBV (1,25). Gloves need not be worn when feeding patients and when wiping saliva from the skin.

Special precautions, however, are recommended for dentistry (1). Occupationally acquired infection with HBV in dental workers has been documented (4), and two possible cases of occupationally acquired HIV infection involving dentists have been reported (5,26). During dental procedures, contamination of saliva with blood is predictable, trauma to health-care workers hands is common, and blood spattering may occur. Infection control precautions for dentistry minimize the potential for nonintact skin and mucous membrane contact of dental health-care workers to blood-contaminated saliva of patients. In addition, the use of gloves for oral examinations and treatment in the dental setting may also protect the patient's oral mucous membranes from exposures to blood, which may occur from breaks in the skin of dental workers' hands.

Use of Protective Barriers

Protective barriers reduce the risk of exposure of the health-care worker's skin or mucous membranes to potentially infective materials. For universal precautions, protective barriers reduce the risk of exposure to blood, body fluids containing visible blood, and other fluids to which universal precautions apply. Examples of protective barriers include gloves, gowns, masks, and protective eyewear. Gloves should reduce the incidence of contamination of hands, but they cannot prevent penetrating injuries due to needles or other sharp instruments. Masks and protective eyewear or face shields should reduce the incidence of contamination of mucous membranes of the mouth, nose, and eyes.

Universal precautions are intended to supplement rather than replace recommendations for routine infection control, such as handwashing and using gloves to prevent gross microbial contamination of hands (27). Because specifying the types of barriers needed for every possible clinical situation is impractical, some judgment must be exercised.

The risk of nosocomial transmission of HIV, HBV, and other bloodborne pathogens can be minimized if health-care workers use the following guidelines:*

1. Take care to prevent injuries when using needles, scalpels, and other sharp instruments or devices; when handling sharp instruments after procedures; when cleaning used instruments; and when disposing of used needles. Do not recap used needles by hand; do not remove used needles from disposable syringes by hand; and do not bend, break, or otherwise

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*The August 1987 publication should be consulted for general information and specific recommendations not addressed in this update.

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CENTERS FOR DISEASE CONTROL January 26, 1990 / Vol. 39 / No. RR-1

MMWR Recommendations and Reports

MORBIDITY AND MORTALITY WEEKLY REPORT

Public Health Service Statement on Management of Occupational Exposure to Human Immunodeficiency Virus, Including Considerations Regarding Zidovudine Postexposure Use

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
Atlanta, Georgia 30333

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was culture-positive for HIV 4 months after completing 6 weeks of chemotherapy. The second person was exposed to a high concentration of HIV on abraded skin in a research laboratory, was started on zidovudine within 24 hours of postexposure, and remains HIV-seronegative after 11 months. The risk of seroconversion after this type of laboratory exposure is unknown. The third person was exposed to a high concentration of HIV on broken skin in a research laboratory, was started on zidovudine within 24 hours after the exposure, and is HIV-seronegative 3 months after the exposure. The risk of

seroconversion after this type of laboratory exposure also is unknown. All individuals were able to complete a 6-week course of therapy (200 mg orally every 4 hours) without clinically significant adverse effects. Information regarding enrollment in this study can be obtained by calling the NIAID study coordinator at (800)537-9978.

Prophylaxis Schedules Currently Used After Occupational Exposure

Various regimens have been prescribed for zidovudine prophylaxis after occupational exposure. No data are available to enable investigators to determine the efficacy or compare the toxicity of these or other regimens. At the National Institutes of Health Clinical Center, workers who elect to receive zidovudine are treated with 200 mg every 4 hours (six times daily) for 6 weeks (6). At San Francisco General Hospital, workers who elect to receive zidovudine are treated with 200 mg every 4 hours (five times daily; not dose is given at 4:00 a.m.) for 4 weeks (6). Some clinicians have used an initial dose of 400 mg, and others have prescribed treatment courses ranging from 4 days to 4 months. At several institutions, attempts are made to begin prophylaxis within 1 hour after exposure for workers who elect to receive the drug.

DISCUSSION

Data from animal and human studies are inadequate to establish the efficacy or safety of zidovudine for prophylaxis after occupational exposure to HIV. However, some physicians believe that zidovudine should be offered as prophylaxis to persons after certain occupational exposures for the following reasons: the severity of the illness that may result from HIV infection, the documented antiviral effect of zidovudine in the treatment of persons with established HIV infection, the apparent reversibility of acute toxicity in persons taking zidovudine for a brief period, and the course of some retroviral infections. Other physicians believe that zidovudine should not be recommended for uninfected persons after occupational exposures because of

the lack of data demonstrating efficacy in postexposure prophylaxis, the limited data on toxicity in uninfected individuals, and the fact that zidovudine has been shown to be carcinogenic in rats and mice.

At this time, prophylaxis with zidovudine cannot be considered a necessary component of postexposure management. However, workers who might be at risk of occupational exposure to HIV should be informed, as part of job orientation and ongoing job training, of the considerations pertaining to the use of zidovudine for postexposure prophylaxis. The PHS recommends that if a physician decides to offer zidovudine to a worker after an exposure incident, that decision by the physician and the decision by the worker to take zidovudine should take into account the following considerations.

Considerations Regarding Use of Zidovudine After an Occupational Exposure

Risk of HIV infection after exposure

Evaluation of the risk of HIV infection after exposure should take into account existing knowledge from prospective studies of exposed workers, which demonstrate that on the average the risk of transmission of HIV per episode of percutaneous exposure (e.g., a needlestick or cut with a sharp object) to HIV-infected blood is approximately 0.4%. These studies also suggest that the risk of HIV transmission per episode of mucous-membrane or skin exposure to HIV-infected blood is less than that after a percutaneous exposure (7,18-21). The risk of HIV transmission after occupational exposure to body fluids other than blood, for which universal precautions are recommended, is unknown. The risk of HIV infection for persons who take zidovudine postexposure prophylaxis cannot be determined at present because of the small number of persons studied.

Risk evaluation should also include an assessment of factors that may increase or decrease the probability of HIV

transmission after an individual occupational exposure. These factors are not well understood, but include the likelihood that the source fluid contained HIV and probably also the concentration of HIV in the source fluid, the route of exposure, and the volume of fluid involved. For example, a percutaneous exposure to concentrated HIV in a research laboratory is probably more likely to result in transmission of infection than a similar exposure to HIV-infected blood in a clinical setting. A percutaneous exposure to HIV-infected blood is probably more likely to result in transmission than a mucous-membrane exposure to the same blood. Finally, an exposure to a larger quantity of HIV-infected blood, such as infection of several milliliters, is probably more likely to result in HIV transmission than an exposure to a smaller quantity of the same blood, such as in a needlestick exposure.

Interval between exposure and initiation of prophylaxis, if given

Data from animal studies suggest that prophylaxis against certain retroviral infections other than HIV may be more effective when started within hours after exposure (22,23). Because in vitro studies indicate that human HIV infection may be established in human lymphocytes within hours after exposure (24), and epidemiologic studies of exposed health-care workers indicate that acute retroviral illness may occur as early as 2 weeks after exposure (7), it appears that if the decision is made to use postexposure prophylaxis, prophylaxis should be initiated promptly.

Counseling and informed consent

If zidovudine prophylaxis is being considered, the worker should be counseled regarding a) the theoretical rationale for postexposure prophylaxis, b) the risk of occupationally acquired HIV infection due to the exposure, c) the limitations of current knowledge of the efficacy of zidovudine when used as postexposure prophylaxis, d) current knowledge of the toxicity of zidovudine (including the data from animal and human studies) and the limitations of this knowledge in predicting

toxicity in uninfected individuals who take the drug after occupational exposures, and e) the need for postexposure follow-up (including HIV serologic testing), regardless of whether zidovudine is taken. The worker should also be informed that there are diverse opinions among physicians regarding the use of zidovudine for postexposure

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Post-Exposure Evaluation and
Follow-Up Requirements under
OSHA's Standard for Occupational
Exposure to Bloodborne Pathogens

A GUIDE TO DENTAL EMPLOYER OBLIGATIONS

Prepared by the American Dental Association in
cooperation with the Occupational Safety and Health
Administration U.S. Department of Labor
December 1997

[Add. 167]

Post-Exposure Evaluation and Follow-Up Requirements under OSHA's Standard for Occupational Exposure to Bloodborne Pathogens
A Guide to Dental Employer Obligations

OVERVIEW

OSHA's final rule for *Occupational Exposure to Bloodborne Pathogens [29 CFR 1910.1030(f)]* requires the dental employer to make immediately available confidential medical evaluation and follow-up to an employee reporting an exposure incident.

An exposure incident is any eye, mouth, mucous membrane, non-intact skin, or other parenteral contact with blood or other potentially infectious material (OPIM). (For example, a puncture from a contaminated sharp such as an injection needle or a cut from a scalpel blade or suture needle.) Saliva in dental procedures is treated as OPIM.

The dental employer must refer the exposed employee to a licensed health care professional. This means a person who is licensed under the laws of the state where he/she practices to independently provide the post-exposure evaluation and follow-up services required by the standard. The health care professional will counsel the individual about what happened and how to prevent further spread of any potential infection. He or she will prescribe appropriate follow-up in accordance with current U.S. Public Health Service recommendations. The licensed health care professional also will evaluate any reported illness to determine if the symptoms may be related to Human Immunodeficiency Virus (HIV) or Hepatitis B Virus (HBV) infection.

Note: The standard is designed to prevent occupational exposure to blood or OPIM. If the required controls are in place and the standard is correctly implemented, then exposure incidents will be relatively uncommon events.

STEP-BY-STEP GUIDE TO COMPLAINCE

Note: This section is designed to supplement the attached flow chart outlining the standard's requirements following an exposure incident.

Reporting Incident—
Employees should immediately report exposure incidents to the employer to permit timely medical follow-up. According to the U.S. Public Health Service, if HIV postexposure prophylaxis is medically indicated it should be initiated promptly, preferably within 1-2 hours after the exposure incident. Immediate reporting also enables the dental employer to evaluate the circumstances surrounding the exposure incident to try to find ways to prevent such a situation from occurring again.

Referral to a Health Care Professional (HCP)—
Following a report of an exposure incident, the dental employer shall make immediately available to the exposed employee a

confidential medical evaluation and follow-up at no cost to the employee. The dental employer is responsible for providing follow-up, but is not required to perform the follow-up. The employer must refer the exposed employee to a licensed health care professional who will perform all medical evaluations and procedures in accordance with the most current recommendations of the U.S. Public Health Service.

Note: *The bloodborne pathogens standard is a performance oriented standard. As such, it requires that medical evaluation and follow-up be provided in accordance with the U.S. Public Health Service recommendations, but does not cite specified recommendations. OSHA intentionally drafted the standard in this fashion to ensure that the most current recommendations would be followed.*

Documentation – The dental employer must prepare a report of the exposure incident, including the route(s) of exposure, the circumstances under which the exposure incident occurred, and the identity of the source patient – **if known**, and if permitted by law (*see Identification and Testing of Source Patient's Blood on page 3*). This report must be placed in the employee's confidential medical record. A copy also must be provided to the evaluating health care professional.

Note: *Additionally, an exposure incident* ***may*** *meet the criteria for OSHA's Recordkeeping Requirements as a "recordable occupational injury." These requirements apply to dental employers with eleven or more employees and require the completion of OSHA forms 200 (Log and Summary of Occupational Injuries and Illnesses) and 101 (Supplemental Record of Occupational Injuries and Illnesses). The criteria for recording under such circumstances include the following:*
1. The incident results in a loss of consciousness, transfer to another job, or a work restriction, or

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Post-Exposure Evaluation and Follow-Up Requirements under OSHA's Standard for Occupational Exposure to Bloodborne Pathogens

Questions and Answers for Dental Employers

Selection of Health Care Professional - May the dental employer select the health care professional?

Yes, it is the dental employer, rather than the employee, who is entitled to select a health care professional to provide the hepatitis B vaccination and post-exposure evaluation and follow-up services required under the standard. Dental employers would be wise to select a health care professional who is familiar with the standard and the recommended post-exposure evaluation and follow-up protocol. The U.S. Public Health Service recommends that when possible, the medical evaluation and follow-up should be implemented in consultation with persons having expertise in antiretroviral therapy and HIV transmission. On this basis, the dental employer should identify, and have on record, a health care professional with appropriate expertise, or a healthcare professional who consults with a person having such expertise.

Employer Obligation Related to CDC Recommendations – *Since the standard incorporates by reference the CDC recommendations for post-exposure prophylaxis, what is the dental employer's obligations when the CDC guidelines are not clear or when the health care professional recommends treatment beyond what is contained in the CDC guidelines?*

Under the standard, it is the dental employer's responsibility, following an exposure incident, to make immediately available to the exposed employee post-exposure prophylaxis, when

medically indicated. The standard requires that post-exposure prophylaxis be provided in accordance with the recommendations of the U.S. Public Health Service, but it does not cite specific recommendations.

OSHA intentionally drafted the standard in this fashion to ensure that the most current recommendations would be followed. Because post-exposure testing and prophylaxis are rapidly changing and developing fields, it must be provided according to recommendations of the U.S. Public Health Service current at the time post-exposure testing and prophylaxis take place.

Currently, for employees who have not received the HBV vaccine series, HBV vaccine (and in some circumstances hepatitis B immune globulin) is to be offered as soon as possible after the exposure incident, but no more than seven days after the incident.

With regard to HIV disease, CDC guidelines for post-exposure antiretroviral drug therapy are constantly evolving. The HCP providing post-exposure evaluation and follow-up must ensure that current CDC guidelines are followed. In order to assist the evaluating HCP in following CDC guidelines, the dental employer must ensure that the exposed employee receives **immediate** post-exposure evaluation (according to the CDC, preferably within 1-2 hours, but not longer than 24 hours) after the exposure incident.

Note: *The recommendations of the U.S. Public Health Service provides guidelines for post-exposure chemoprophylaxis. However, the use of post-exposure chemoprophylaxis is a clinical decision which should be individualized for each employee incident.*

Changes to CDC Recommendations - *How will dentists know of changes in the CDC guidelines that affect their OSHA obligations?*

CDC guidelines and recommendations are widely distributed and readily available to health care professionals either directly from the CDC or through professional associates. Direct subscriptions to the Morbidity and Mortality Weekly Report (MMWR) are available. Due to the ever-changing nature of the health care industry, professionals routinely seek to keep themselves abreast of new de-

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[Annals of Plastic Surgery Vol. 29/No.2/August 1992]

The Use of a Contained Breathing Apparatus to Isolate the Operator and Assistant for Aerosolizing Procedures Including Dermabrasion and Laser Surgery

Paul J. Weber, MD
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[Figures and References omitted]

Dermabrasion and carbon dioxide laser surgery aerosolize patient blood and tissue particles. The operating physician and assistants may therefore have intimate inhalational and mucous membrane contact with patients' body fluids. Herein is described the use of an isolated ventilation system to protect physicians and assistants from blood and tissue products aerosolized during the course of dermabrasion and carbon dioxide laser surgery.

Weber PJ, Wulc AE: The use of a contained breathing apparatus to isolate the operator and assistant for aerosolizing procedures including dermabrasion and laser surgery. Ann Plast Surg 1992;29:182-184

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The authors have absolutely no financial or other connection with Gast Corporation, manufacturer of the REGENAIR, or with any other company that makes any product remotely related to the scope of this paper.

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Traditionally performed, dermabrasion and carbon dioxide laser surgery unfortunately provide the operating physician and assistants with intimate inhalational and mucous membrane contact with patients' body fluids. Presently, there is much

concern regarding human immunodeficiency virus (HIV) infection and body fluids safety. This concern is enhanced by an imperfect ability to detect HIV infection in some individuals. Herein is described the use of an isolated ventilation system to protect physicians and assistants from patients' blood and tissue products aerosolized during the course of dermabrasion and laser surgery.

Material and Methods

The electrically operated air compressor (trademarked as the REGENAIR, manufactured by Gast Corporation of Benton Harbor, MI) provides up to 3 kg/cm^2 of air pressure. The REGENAIR has a maximal air-flow rate of 0.25 m^3/min. Because this breathing apparatus is especially made for deep underwater use, there is no chance of operating-room air leakage onto the face while the compressor is functioning. Two masks and 15 m of hose are included with the air-compression unit. The masks cover the entire face (including spectacles) with an airtight seal. A constant bath of fresh air is blown over the face and expelled through a one-way valve. This air provided to the surgeon and assistants is "fresh" if the compressor (air source) is placed in a room down a hall, the hoses pass 15 m down the hall, and the operating room door is closed (except for a 4 x 2-cm hole cut in the outer bottom corner for hose passage).

Ten full-face dermabrasions and nine carbon dioxide laser ablative procedures have been performed using this apparatus. The mask is surprisingly easy to apply and remove, and does not interfere with the dermabrasion or laser procedures in the least, with the exception of the need to talk loudly over the rush of air. Important points include the following: Schedule individual patients at the end of the day. When work on a patient is completed, leave the air flowing; do not remove the masks. If available, open the windows to the operating room, escort the patient into another room for dressings, close the operating room door, and then, once safely in a separate room, remove the masks.

Discussion

Dermabrasion and carbon dioxide laser surgery are two surgical techniques that may result in aerosolized patient tissue and debris. Numerous adjuncts have been described to aid in the per-

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formance and to enhance the safety of dermabrasion and laser surgery. However, to our knowledge, no adjuncts have been described that eliminate the possibility of respiratory and mucous membrane exposure to patients' blood and tissue microdroplets. Herein is described the first use of an isolated ventilation system for the performance of dermabrasion and laser surgery.

Recently, studies have shown that portions of viruses or whole viruses may become airborne during laser surgery. Sawchuck and co-workers were the first to detect viable bovine papilloma virus obtained from the air surrounding carbon dioxide laser ablation sites. A great concern in dermabrasion, a procedure in which microdroplets of blood and tissue are cast into the surrounding air, is the possible dissemination of HIV. The detection of antibodies to HIV is not as sensitive as the detection of HIV antigen, and antigen detection is not as sensitive as HIV culture. Unfortunately, the most practical preoperative test may be the HIV-EI (enzyme immunoassay). Some surgeons require an HIV-EI before dermabrasion and carbon dioxide laser ablative procedures. However, there is a "window period," in which an HIV carrier may not be detected by the enzyme immunoassay test. This problem may be especially concerning because of the relatively long duration of the enzyme immunoassay window period which may range between several weeks and 6 months. This window period may explain cases of transfusion-associated HIV transmission that have occurred since the institution of HIV type 1 antibody testing in early 1985.

Because approximately 1 million of 240 million persons residing in the United States are HIV positive, a surgeon who performs 100 dermabrasions or carbon dioxide laser procedures over 5 years may be at some risk for inhaling or encountering HIV. Therefore, HIV, traditionally a physical contact and a bloodborne disorder, may through dermabrasion become an airborne disorder.

Traditional dermabrasion and carbon dioxide laser ablative surgical wear includes glasses, surgical mask, Plexiglas welder's hood, surgical hair cap, full surgical gown, and gloves. Although the welder's hood stops the direct flight of particles, the operating room air may be laden with particulate patient tissue. Millions of these airborne particles surround and bathe the operating phy-

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sician and assistants. These airborne particles can flow past or around the imperfect seals of the aforementioned traditional "barriers." Once past these traditional barriers, including the surgical masks, the particles may encounter the mucous membranes or respiratory systems. Therefore, danger exists. Some previously published experiments skirt this problem. For example, Sawchuck and co-workers imply that surgical masks offer protection against airborne papilloma virus. A major qualification should be that the mask be perfectly sealed to the face as its periphery. The conditions of the referenced experiment regarding the mask filter reveal that a truly occlusive peripheral seal had been made by using a tight rubber band placed over a special collecting device. Unfortunately, virtually all surgical masks do not provide a perfect seal under real operating room conditions.

The entire REGENAIR unit retails for approximately $1000. Herein is described the use of an isolated ventilation system to

protect physicians and assistants from blood and tissue products aerosolized during the course of dermabrasion and carbon dioxide laser surgery.

[References Omitted]

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INTERROGATORY NUMBER 5

Please state whether your intention in approaching Dr. Bragdon for dental treatment was to obtain services or whether you were motivated in part or in whole by a desire to obtain an order against Dr. Bragdon for violation of the Maine Human Rights Act or the Americans with Disabilities Act.

RESPONSE TO INTERROGATORY NUMBER 5

My intention in making an appointment with Dr. Bragdon was to obtain dental treatment for a tooth ache. I was in need of dental treatment and if Dr. Bragdon had agreed to provide dental services to me, I would have accepted treatment from him. I understood that Dr. Bragdon might turn me away because I was HIV-positive and if he did, I planned to take legal action in order to make him comply with the law.

INTERROGATORY NUMBER 6

If your action in obtaining an appointment with Dr. Bragdon was in any way motivated by a desire to obtain an order under the Maine Human Rights Act or the Americans With Disabilities Act, please describe in detail all of the circumstances leading to your appointment, including identifying any individuals with whom you consulted prior to making the appointment.

RESPONSE TO INTERROGATORY NUMBER 6

OBJECTION: Ms. Abbott objects to Interrogatory Number 6 because the wording implies that by answering this Interrogatory, Ms. Abbott would be affirming the statement in Interrogatory Number 6 that she obtained an appointment with Dr. Bragdon due to a "desire to obtain an order" against him. Ms. Abbott does not adopt as her own statement any of the wording in Interrogatory Number 6. Without waiving this objection, Ms. Abbott answers Interrogatory Number 6 as follows:

In August, 1994, I had tooth pain. Sally Lou Patterson,

then Executive Director of the Eastern Maine AIDS Network (EMAN), recommended that I see a dentist named Dr. Cassa at the Westgate Dental office in Bangor, Maine.

My first appointment with Dr. Cassa was on or about August 8, 1994. Dr. Cassa's staff cleaned my teeth. My second appointment with Dr. Cassa [was] on or about August 17, 1994.

Dr. Cassa did a dental exam and took x-rays of my mouth. My third appointment with Dr. Cassa was on or about August 25, 1994, at which, as is explained below, he performed a root canal without telling me until after the procedure was over.

I was extremely dissatisfied with Dr. Cassa's care. During my three visits, Dr. Cassa did not discuss with me my dental condition or the dental treatment and procedures he was performing. For example, on or about August 25, 1994, Dr. Cassa did an extensive dental procedure, but did not tell me until it was over that the problem was "worse than he thought" and that he had just performed a root canal. I was just as disturbed by the arguing and fighting between Dr. Cassa and his assistant while they were treating me. This included arguing and struggling while they were attempting to place a dental dam on my tooth.

At the end of the August 25 session, Dr. Cassa indicated that I had additional dental work which needed to be done. I made an appointment for September 19, 1994, but felt strongly that I did not want to return to Dr. Cassa.

I told Sally Lou Patterson about my experience with Dr. Cassa and that I did not want to return to him. Sally Lou

Patterson later asked if I would be willing to go to Dr. Bragdon for dental treatment. She mentioned that Dr. Bragdon was the dentist whom Doug Worster-Jones had the lawsuit against. Sally Lou Patterson gave me Dr. Bragdon's telephone number and I told her that I would make an appointment with him. I understood that if Dr. Bragdon would not treat me, I would be taking legal action against him.

INTERROGATORY NUMBER 7

Describe in detail the basis for your allegation in paragraph 14B of your complaint that Dr. Bragdon has imposed eligibility criteria, for the privilege or advantage of receiving dental services, to screen out the class of persons who have tested positive for HIV in violation of 14 U.S.C. Section 12182(b) (2) (A) (i).

RESPONSE TO INTERROGATORY NUMBER 7

OBJECTION: Ms. Abbott objects to Interrogatory Number 7 because it calls for a legal conclusion and because the phrase "describe in detail" is vague and unclear. Without

waiving this objection, Ms. Abbott directs the defendant to Dr. Bragdon's Infectious Disease Policy.

INTERROGATORY NUMBER 8

Please describe in detail what reasonable modifications in policies, practices, or procedures or where such modifications are necessary to afford equal advantages or privileges to the class of persons who have tested positive for HIV, in accordance with 42 U.S.C. Section 12182 (b) (2) (A) (ii) Dr. Bragdon should make.

RESPONSE TO INTERROGATORY NUMBER 8

OBJECTION: Ms. Abbott objects to Interrogatory Number 8 because it calls for a legal conclusion. Without waiving this objection, Ms. Abbott states that Dr. Bragdon needs to modify his

***

AMERICAN JOURNAL OF NURSING

TB PLUS HIV

By Lynn M. O'Brien and Kathleen A. Bartlett

[References omitted]

The HIV positive are more prone not only to contract deadly *Mycobacterium tuberculosis* but to transmit it in a drug-resistant form—and it evades diagnosis.

Until recently, scientists and clinicians had hoped to eradicate tuberculosis nationwide by the year 2010. Since 1986, however, the number of cases reported has continued to rise. In 1990, 25,701 cases were reported - an increase of 9.4% over 1989 and the largest annual rise since 1953. Of particular concern is the appearance of both *Mycobacterium tuberculosis* (MTB) and isoniazid (INH)-resistant MTB in people who are HIV positive. Moreover, as the disease appears more frequently in out patients, health care workers are more likely to acquire it themselves.

First, dust off the basics

Mycobacterium tuberculosis is an acid fast, acrobic bacterium. It is most commonly found in particles known as droplet nuclei. These droplet nuclei become airborne when a person with pulmonary or laryngeal tuberculosis coughs, sneezes, speaks or sings. The bacilli are then inhaled and most often lodge in the upper portion of the lung and spread throughout the body of a susceptible host.

Primary infection with MTB most often occurs within one or two years of exposure. Those most susceptible are children

(whose immune systems are not fully developed), the aged (whose immune systems are diminished), under or malnourished people, residents of overcrowded living quarters, and those with an immune system compromised by illness. Old infection can reactivate in those persons who initially mounted an immune response and walled off the bacteria, but in whom the bacilli can now resume their multiplication due to a defective or deficient immune system.

Thus, the HIV-positive patient with a deficient immune system has a much higher risk of developing either primary disease or reactivation of old disease. It also appears that the time between exposure and development of active tuberculosis is shorter in HIV-positive patients than in other people.

What if the skin test is negative?

People who have an HIV infection and a low CD4 cell count may not be able to mount an immune response to a tuberculin skin test, so a negative PPD skin test cannot be relied upon. The likelihood of a falsely negative tuberculin skin test rises as the stage of HIV infection progresses. (Even in the immunocompetent, PPD skin tests should be considered only as a screening test, not as a definitive diagnostic tool.)

To further complicate detection, acid-fast sputum smears may be negative. Less commonly, even chest X-rays may be equivocal and the chest and lungs may appear to be normal on physical exam. Systemic symptoms such as weight loss, cough and night sweats, may be thought due to another opportunistic disease. Finally, pulmonary tuberculosis may be associated with disease at extrapulmonary sites, a phenomenon not usually seen in those with normal immune systems.

Unfortunately, patients who are HIV positive and become infected with MTB may experience a higher frequency of side effects from traditional TB medicines. These side effects may lead them to skip doses or to stop taking the drugs, which in turn may allow the MTB organisms to develop resistance to INH. Every person who develops drug resistance thus raises the risk of transmission of a resistant strain to other susceptible hosts. TB

drugs may interact with the drugs being used to treat the fungal in-

***

Lynn M. O'Brien, RN, MS, is a certified nurse practitioner in the infectious disease clinic, Veterans Medical Center, San Francisco, CA, where she provides primary care to HIV-infected patients and screens for and treats those with tuberculosis. Kathleen A. Bartlett, RSN, is a public health nurse who coordinates the same clinic. She also tracks and reports infectious diseases in this VA's outpatients.

[Add. 176]

fections common in HIV-positive patients. The antifungal drugs may reduce the absorption of INH or rifampin; this poorer absorption may also lead to drug-resistant organisms. Yet, it has been suggested that HIV-infected patients may require a longer regimen of drug treatment for MTB.

At the San Francisco Veterans Affairs Medical Center, most HIV-infected patients are followed by the infectious disease service, which includes two infectious disease clinics, an aerosolized pentamidine clinic, a nurse practitioner-run drop-in clinic and various nurse-run research clinics. Often, the patients' first and most frequent contact thereafter is a nurse or a nurse practitioner. In addition, hospitalized patients receive the bulk of their care from the hospital nursing staff. Nurses are often involved in the diagnosis and care of those infected with MTB, and nurses are often the people who devise and enforce methods to prevent transmission to other patients or staff. We urge our colleagues to consider the possibility of MTB in any HIV-positive patient who develops cough, fever, and weight loss. In our clinic, it is a nursing responsibility to alert the patient's physician, or to initiate the TB workup ourselves.

Searching for elusive mycobacteria

The TB workup begins with a history of the patient's past or recent exposure. A physical exam of the chest/lungs follows,

including auscultation and percussion. The patient is given a tuberculin skin test, keeping in mind that a person whose CD4 count is below 200 may no longer show an immune response to the PPD antigen. Thus, at the time of the PPD injection, HIV-infected patients are also given a skin test for anergy. If this intracutaneous injection of *Candida* and mumps antigens in the opposite arm produces an immune response (induration at the injection site), then we can be fairly sure that the patient is able to produce an immune response to the tuberculin, as well, if he has been infected.

The response to the TB skin test is considered positive if the induration is 5 millimeters or larger, instead of the usual positive response of 10 mm or larger in other high-risk groups, or 15 mm or larger in otherwise healthy people.

As the adage says, however, "absence of proof does not prove absence of disease." So a chest X-ray with PA and lateral views is obtained and read both by the radiologist and the patient's physician. The X-ray might reveal infiltrates in the upper lung fields, hilar adenopathy, pleural effusion, a miliary pattern of cavitation. Even so, hilar adenopathy and a miliary pattern may be mistaken as evidence of other opportunistic diseases, such as *Pneumocyst carinii* pneumonia, Kaposi's sarcoma or lymphoma. A small fraction of chest X-rays may even be normal despite pulmonary tuberculosis infection.

Often the last—yet perhaps the most critical—step in the MT

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[Add. 177]

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

NO. 96-1643

SIDNEY ABBOTT, ET AL.,

Plaintiffs-Appellees,

VS.

RANDON BRAGDON, DMD,

Defendant-Appellant

ON APPEAL FROM A JUDGMENT OF
THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MAINE

BRIEF OF THE AMICI CURIAE RHODE ISLAND DEPARTMENT OF PUBLIC HEALTH, DEPARTMENT OF PUBLIC HEALTH OF THE COMMONWEALTH OF MASSACHUSETTS, BUREAU OF HEALTH OF THE MAINE DEPARTMENT OF HUMAN SERVICES, AMERICAN PUBLIC HEALTH ASSOCIATION, ASSOCIATION OF STATE AND TERRITORIAL DENTAL DIRECTORS, AND ASSOCIATION OF STATE AND TERRITORIAL HEALTH OFFICIALS.

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For Amicus Curiae

Department of Public Health
Commonwealth of Massachusetts

[Add. 178]

No. 97-156

In The

Supreme Court of the United States
October Term, 1997

RANDON BRAGDON, D.M.D.

Petitioner,

-v.-

SIDNEY ABBOTT, et al.,

Respondents.

ON WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE FIRST CIRCUIT

BRIEF OF INFECTIOUS DISEASES SOCIETY OF AMERICA; NATIONAL ALLIANCE OF STATE AND TERRITORIAL AIDS DIRECTORS; ASSOCIATION OF STATE AND TERRITORIAL DENTAL DIRECTORS; AMERICAN ASSOCIATION OF DENTAL SCHOOLS; COUNCIL OF STATE AND TERRITORIAL EPIDEMIOLOGISTS; AMERICAN NURSES ASSOCIATION; ASSOCIATION OF NURSES IN AIDS CARE; NATIONAL ASSOCIATION OF PUBLIC HOSPITALS AND HEALTH SYSTEMS; AMERICAN PUBLIC HEALTH ASSOCIATION; ASSOCIATION OF REPRODUCTIVE HEALTH PROFESSIONALS ; JOHN G. BARTLETT, M.D.; JAMES W. CURRAN, M.D., M.P.H.; JOHN C. FLETCHER, PH.D., M. DIV.; JUNE E. OSBORN, M.D.; MERLE A. SANDE, M.D.; BRIAN WISPELWEY, M.S., M.D. AS *AMICI CURIAE* IN SUPPORT OF RESPONDENT

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[Add. 179]

No. 97-156

In The
SUPREME COURT OF THE UNITED STATES
October Term, 1997

RANDON BRAGDON, D.M.D., *Petitioner*,

v.

SIDNEY ABBOTT, ET AL., *Respondents*.

ON WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE FIRST CIRCUIT

BRIEF OF THE AIDS ACTION COUNCIL, AIDS NATIONAL INTERFAITH NETWORK, AMERICAN ASSOCIATION ON MENTAL RETARDATION, AMERICAN ASSOCIATION OF RETIRED PERSONS, AMERICAN CIVIL LIBERTIES UNION, AMERICAN COUNCIL OF THE BLIND, AMERICAN FOUNDATION FOR AIDS RESEARCH, *ET AL.*, AS *AMICI CURIAE* SUPPORTING RESPONDENTS

[Additional *Amici Curiae* Listed Inside Cover]

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February 1998 *Counsel of Amici Curiae*

[Add. 180]

No. 97-156

In The
SUPREME COURT OF THE UNITED STATES
October Term, 1997

RANDON BRAGDON, D.M.D.,
Petitioner,

v.

SIDNEY ABBOTT, *et al.*,
Respondents.

On Writ of Certiorari to the
United States Court of Appeals
for the First Circuit

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[Add. 181]

THE BODY: AN
AIDS AND HIV
INFORMATION
RESOURCE

IAPAC

How Far Can You Knock Down HIV?

An Interview with David D. Ho., MD

by Mark Mascolini

- Introduction
- The primary infection experiment
- Not just "math and modeling"
- Thinking ahead with anti-HIV regimens
- "Not a time to pull back" on quest for new drugs
- The eradication wager
- References

When attempting to write a pithy preamble to an interview with David Ho, one is tempted to consult the latest edition of The Toastmaster's Handbook for snappy alternatives to the much-abused "Our speaker tonight needs no introduction." Even before the December 30, 1996 issue of Time, telling this Journal's readers something new about the director of the Aaron Diamond AIDS Research Center would have been difficult.

But perhaps there is a way to illumine further his contribution to HIV research, and that is to announce here that the Journal has selected Ho as the first recipient of what is certain to become a highly coveted prize: Author of the Most-Stolen Slide of the Year award. In fact, if the Journal had begun bestowing this

trophy a few years back, Ho would now have at least three platinum AMOSSYs festooning his office. The first would commemorate the celebrated image from his 1989 New England Journal article demonstrating astoundingly high titers of HIV in plasma and PBMCs of seropositive people with and without AIDS-defining symptoms. The second would show plummeting levels of plasma RNA in people whose steady state of viral replication had just been perturbed by a protease inhibitor, evidence that led to the

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http://www.thebody.com/iapac/ho.html 8/18/98

[Add. 182]

IAPAC: Interview with David Ho, MD

[page 3]

Journal: ***You've said you want to continue treatment for two and a half to three years before you stop. When you do stop, how long would it take for you to be convinced that the therapy had worked and had eradicated the virus?***

Ho: We're in uncharted territory here. We really don't know, but we have the feeling from previous observations, when therapy was stopped earlier, that when HIV returns it returns quickly. We plan to monitor the patients very, very frequently and carefully, and I think we will feel pretty good if we don't see any virus in the first month or two. Obviously, there could be a late appearance, but knowing how quickly HIV doubles, we really should see detectable levels of virus within days or weeks – unless there's something we don't understand. In that case there could be occurrences months or years later. Obviously, we will continue to follow these patients very carefully even if HIV can't be detected in the first few months after treatment stops.

Journal: ***Let's say that it doesn't work and virus isn't eradicated in these studies. Based on what's known now, are there still good reasons to start treating people with primary infection, even if you can't clear the virus?***

Ho: The only patient data to justify intervention during primary infection is the study by Perrin and his colleagues in Geneva. They showed that, over the short term, AZT treatment of primary infection has some clinical benefit compared with placebo. Beyond that, I don't think there's any real information to guide us. I think we have to rely on our current understanding of what HIV does and what the drugs do in other settings: since there's now growing evidence that multidrug combination therapy is better than monotherapy or dual therapy, and since we know we get the best viral response when the patient is treated earlier, one could extend that logic to the primary setting. But that's only a theoretical argument rather than something that's supported by clinical data at this point.

Journal: ***You've been careful to say that you think of these studies as proof-of-concept studies rather than as an attempt to establish a standard of care. But what exactly does that mean? There are some clinicians who already have adopted this strategy as the standard of care for primary HIV infection. Do you think they shouldn't until your results and those of others are more mature?***

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Http://www.thebody.com/iapac/ho.html 8/18/98

[Add. 183]

USA TODAY●WEDNESDAY, SEPTEMBER 16, 1998●3A
THE NATION

Scientists solve mystery of Jupiter's rings

[Picture omitted]

Pictures suggest asteroid impacts stirred moon dust

By Dennis Cauchon
USA TODAY

Astronomers have discovered why the giant planet Jupiter has rings around it – and the answer surprised even the experts.

Using photos from the Galileo spacecraft now circling the solar system's largest planet, scientists determined that Jupiter's three rings are made of moon dust. The dust was kicked up over millions of years as asteroids and comets crashed into four small moons near Jupiter's surface.

The discovery is especially important for two reasons, according to scientists at Cornell University and the National Optical Astronomical Observatories:

- It goes a long way toward explaining all the rings around Neptune and Uranus and some of the rings around Saturn.
- That Jupiter's rings are made up of moon dust is one of the few things about the solar system's origin that scientists believe they know with certainty.

"Definitive results are hard to get in science. Usually, the evidence is circumstantial. But in this case, we really know," said Michael Belton, head of the Galileo imaging team.

The Galileo team solved the mystery by delicately turning the 5,000-pound spacecraft, which was launched by the space shuttle in 1989, so as it passed through Jupiter's shadow, it faced the rings. During this several hours of shade in November 1996, Galileo saw the rings lighted by the sun, providing unprecedented details of the tiny dust particles.

On Earth, the astronomers were surprised to see the three rings stopped abruptly at different moons.

Belton said he thought at first that the data was faulty.

Astronomer Joseph Burns, the Galileo team's ring expert, confessed that he initially had no idea why the rings stopped at moons.

Burns figured out the answer on a plane returning to Cornell in Ithaca, N.Y., where he teaches, from Galileo's headquarters at the Jet Propulsion Laboratory in Pasadena, Calif.

"My first thought was, 'Can I get off this plane? I've got it!' " Burns recalled.

A second flyby in Jupiter's shade in September 1997 confirmed the astronomers' theories: The depths of the three rings matched the depths of the moons' orbits, and the material in the rings matched the dark red dust on the moons' surfaces.

Previously, scientists had thought the rings were caused by interplanetary volcanic dust from Io, a bigger, more distant moon.

Instead, the dust came from the four small moons closest to Jupiter's surface: Metis, Adrastea, Amalthea and Thebe, until the Voyager spacecraft passed by in 1979. There are more moons than rings, the scientists say, because some of the moons supply dust to more than one ring.

The four moons are just 12 to 120 miles wide, and they get pounded by debris. Every hour or so, a sand-sized particle hits the moons. Every few million years, an object as wide as a half-mile leave a big crater.

These objects strike the moons at a speed 100 times that of a .22 bullet. "It's like shooting a bullet into sand: the sand bursts up, and because there's little gravity, it escapes into the ring," said Maureen Ocker-Bell, a Galileo researcher.

The dust specs are few and far between, perhaps one every 30 yards.

The solar system's most famous rings, the inner rings around Saturn, probably have a different origin than the ones discovered at Jupiter, astronomer Joseph Veverka said. Those rings include snowball-sized ice chunks, he said. However, Saturn's less visible outer rings could be similar in origin to Jupiter's.

The Cassini spacecraft, launched in November 1997, will examine those rings when it arrives at Saturn in 2004.

Galileo is studying Europa, another moon of Jupiter, where some scientists suspect there is a frozen ocean that could harbor life.

[Add. 184]

[Rudman & Winchell letterhead omitted]

August 24, 1998

VIA TELECOPIER - ORIGINAL TO FOLLOW

Mark A. Rubin, Esq.
Legal Department
American Dental Association
211 East Chicago Avenue
Chicago, IL 60611

Re: **Sidney Abbott v. Randon Bragdon, D.M.D.**

Dear Mark:

Thank you for letting us know the American Dental Association ("ADA") has decided to file an amicus brief in opposition to Dr. Bragdon on the subject of the ADA's 1991 policy statement on HIV. In fairness to Dr. Bragdon and other members of the ADA who support him, we request that you send us copies of the following materials at your earliest convenience:

1. All committee reports (minority or otherwise), minutes, recommendations, memoranda, etc., generated or having to do with the development of the ADA's 1991 policy statement on HIV (or its reassessment on or before September 16, 1994) by the ADA Council on Science (or similar committee).

2. Materials identifying the "current scientific and epidemiologic evidence" referred to in the ADA's 1991 policy statement on HIV.

3. All documents indicating any controversy among members of the ADA concerning the wisdom of the 1991 policy on HIV.

4. All committee reports (minority or otherwise), minutes, recommendations, memoranda, etc., having to do with the passage by the ADA's House of Delegates of the ADA's 1991 policy statement on HIV, including the 1991 transaction book of the House of Delegates.

[Add. 185]

Mark Rubin, Esq.
August 24, 1998
Page 2

5. The names of the people on the ADA Council on Science (or similar committee) during the development of the ADA's 1991 policy statement on HIV (or its reassessment on or before September 16, 1994), and a place or phone number where they can be contacted.

6. The names of the staff persons of the ADA, and any other persons retained by the ADA, who assisted the ADA Council on Science (or similar committee) on the development of the ADA's 1991 policy statement on HIV (or its reassessment on or before September 16, 1994), and a place or phone number where they can be contacted.

Time is of the essence in this matter, as I am sure you understand. We would therefore appreciate obtaining these materials as soon as possible. If you are unable to respond to this request, or parts of it, it is important that you let us know by August 31, 1998.

We therefore look forward to receiving materials from you or hearing from you concerning this request on or before August 31, 1998.

Thank you very much.

Very truly yours,

/s/ John W. McCarthy
JWM/pal
5300/26606
55999-1
bc: Randon Bragdon, D.M.D.
Charles E. Gilbert, III, Esq.

[Add 186]

[Rudman & Winchell letterhead omitted]

September 9, 1998

VIAL TELECOPIER - ORIGINAL TO FOLLOW

Mark A. Rubin, Esq.
Legal Department
American Dental Association
211 East Chicago Avenue
Chicago, IL 60611

Re: **Sidney Abbott v. Randon Bragdon, D.M.D.**

Dear Mark:

In my letter to you of August 24, I requested copies of materials in this matter. Enclosed for your convenience is a copy of that letter. As I mentioned to you then, time is of the essence; therefore, I had asked that you provide us with these materials by no later than August 31, and/or to contact me by that date if there was any part of the request to which you were unable to respond. As we have not yet received the requested materials nor heard from you, would you let me know as quickly as possible the status of our request.

Thank you for your assistance.

Very truly yours,

John W. McCarthy
JWM/pal
Enclosure
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jmccarthy@rudman-winchell.com

[American Dental Association letterhead omitted]

September 10, 1998

John W. McCarthy, Esq.
Rudman & Winchell
84 Harlow Street
P.O. Box 1401
Bangor, ME 04402-1401

Re: Sidney Abbott v. Randon Bragdon, D.M.D.
United States Court of Appeals-First Circuit
No 96-1643
On remand from the United States Supreme Court

Dear Mr. McCarthy:

This letter is to confirm Dr. Randon Bragdon's consent to the American Dental Association's filing of an *amicus curiae* brief tot he United States Court of Appeals for the First Circuit. Please return a signed original of this letter to me at your earliest convenience in the enclosed self-addressed envelope.

Sincerely,

/s/ Jill A. Wolowitz
Staff Attorney

Accepted and Agreed:

_____
John W. McCarthy, Esq.

JAW/mg
cc: Peter M. Sfikas, Esq., general counsel and associate director,
Division of Legal Affairs, American Dental Association

Mark S. Rubin, Esq. associate general counsel, Division of Legal Affairs, American Dental Association

[Add. 188]

[Rudman & Winchell letterhead omitted]

September 18, 1998

Sent by Telecopy

Jill A. Wolowitz, Esq.
American Dental Association
211 East Chicago Avenue
Chicago, IL 60611-2678

Re: **Sidney Abbott v. Randon Bragdon, D.M.D.**

Dear Ms. Wolowitz:

I have your letter of September 10, 1998, which arrived in my office on September 17, 1998. I am retaining it in my file awaiting the American Dental Association's response to my letters dated August 24, 1998 and September 9, 1998.

Our acquiescence in the *amicus* brief is not dependent upon the content of your response. What we insist on is a response which indicates either that you are honoring our request or that for whatever reason you indicate you are refusing to do so.

Thank you for your assistance.

Sincerely,

/s/John W. McCarthy

JWM/vrs
cc: Mark S. Rubin, Esq.

[Add. 189]

[American Dental Association letterhead omitted]

September 18, 1998

VIA FAX & U.S. MAIL

John W. McCarthy, Esq.
Rudman & Winchell, LLC
The Graham Building
84 Harlow Street
Bangor, ME 04401

Dear Mr. McCarthy:

This is in response to your August 24, 1998 and September 9, 1998 letters to Mark Rubin, on behalf of Dr. Randon Bragdon, for information about the Association's 1991 policy statement on HIV. The 4 volumes of 1991 Transactions are available at a cost of $20.00. Please advise me if this is what you desire.

The balance of your request amounts to discovery of the Association, beyond the scope of a reasonable member request for information, e.g., your request for historical research into reports that may have never existed, for names and phone numbers of former staff, etc. We understand that your Motion has not been acted upon by the Court. In any event, even if we were prepared to address the broad scope of your request, the briefing schedule set by the First Circuit does not give us the time to do so.

Sincerely,

/s/ Peter M. Sfikas
General Counsel and Associate Executive Director
Division of Legal Affairs

PMS:es
cc: John E. Carnes, Esq., Maine Human Rights Commission
Bennett H. Klein, Esq., G.L.A.D.
Jessica Dunsay Silver, Esq., U.S. Department of Justice

[Add. 190]

***

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Printed in the United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2 1

[Add 191]

***

These include, but not (sic) are not limited to, hepatitis B virus (HBV) and human immunodeficiency virus (HIV), which is the etiologic agent for AIDS.

These diseases are not transmitted by casual contact, such as shaking hands. The intact skin is an excellent protective barrier. It is when this barrier is breached by a cut or scrape that there is danger.

There is risk of transmission of these diseases through a needlestick injury or a cut caused by a contaminated instrument. There is danger here because your blood may now come into contact with the infected blood.

Droplet Infection

This type of infection is transmitted by the numerous droplets of moisture, containing bacteria or viruses, that are spread as people talk, breathe, sneeze, or cough. When someone sneezes, he sprays contaminated particles out about 8 feet and up about 4 feet.

Of special concern to the dentist and assistant is the mist of bacteria and debris that is produced by the high-speed handpiece with water spray (Figure omitted).

Being exposed to this mist is approximately the equivalent of having someone sneeze in your face twice per minute—at a distance of 1 foot.

Indirect Transmission

In the dental office, diseases may be indirectly transmitted by soiled hands and towels, dirty instruments, and even dust.

Also, anything that is touched during patient care is considered **contaminated** and potentially capable of spreading disease through indirect contact. (As used here, *contaminated* means something that may possibly have come into contact with a pathogen.)

This includes instruments, faucet handles, switches, handpieces, drawer handles, medications, dressings, the patient's chart, and even the pen used to make the chart entry.

Self-Infection

In many cases, infective microorganisms are present in the patient's mouth but do not cause infection until they enter the bloodstream. However, an open wound in dental surgery may allow these microorganisms access to the bloodstream, and in this way the patient may actually infect himself.

Operator Infection

Infection from the dentist's or assistant's nose, mouth, or hands maybe spread to the patient via droplet infection or by indirect transmission during the operative procedure.

Also, infectious organisms sprayed from the patient's mouth can be transmitted to the dentist or assistant through his or her own nose or mouth or through a break in the skin.

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[Add. 192]

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[Add. 193]

- **Infection Control**

An **infectious disease** is one that is capable of being transmitted with or without actual contact between individuals. The infectious diseases of primary concern in the dental practice are hepatitis, human immunodeficiency virus (HIV), tuberculosis, herpes-viruses, and infectious respiratory agents.

Body Defenses Against Diseases

Intact skin, or skin and mucous membranes that are not broken in any way, acts as a natural protective barrier. **Nonintact skin**, in which there is a cut, scrape, or needlestick injury, does not protect against pathogens entering the body.

Immunity is a condition in which the individual is resistant to a *specific* infection. An individual may be born with immunity to a disease, is immune after having had the disease, or acquires immunity through vaccination.

Vaccinations, also referred to as immunizations, are a major factor in disease prevention. It is extremely important that dental healthcare workers (DHCWs) be immunized against vaccine-preventable diseases. These include

- Hepatitis B
- Influenza
- Measles
- Mumps
- Rubella
- Tetanus

Some of these immunizations, such as that for tetanus, require periodic boosters. To provide adequate protection, immunizations must be kept up to date at all times.

The Chain of Infection

The **chain of infection** describes the four factors that *must* be present before an individual becomes ill with a disease (figure omitted). These factors are

- Virulence
- Numbers
- Susceptible host
- A portal of entry

Virulence. Virulence is the ability of the pathogen to overcome the body's defenses and cause disease. A highly virulent pathogen is able to overcome the body's defenses, even in healthy individuals who are able to resist other diseases.

Numbers. Pathogens must be present in sufficient quantities to overwhelm the body's defenses and produce disease. The number of pathogens may be directly related to the bioburden present. (**Bioburden** refers to organic by-products that may contaminate the surface of an object.)

Susceptible Host. A susceptible host is one who is unable to resist infection by the pathogen. (The **host** is the individual who is ill because of a pathogen.)

Resistance is lowered in an individual who is in poor health, is chronically fatigued, is under extreme stress, and/or has a compromised (damaged) immune system. There are many reasons why the immune system may not function properly. These conditions include HIV infection, immunosuppression therapy (to prevent rejection of a transplanted organ), and radiation or chemotherapy to treat cancer.

Portal of Entry. The pathogens must find an appropriate portal of entry into the body. The portal of entry is specific to each mode of transmission. (**Portal of entry** means door or method of entering the body.)

Disease Transmission

Before it is possible to prevent disease transmission, it is important to understand how it takes place. Table 5-1 summarizes the specialized disease transmission threats that are present in the dental setting.

Bloodborne Transmission

Bloodborne diseases include, but are not limited to, hepatitis B virus (HBV) and HIV. The portal of entry for bloodborne diseases occurs (1) when there is bleeding (bleeding indicates a disruption of the blood stream), (2) when there are breaks in the skin, and (3) by accidental injection through the skin.

Bloodborne transmission, also know as *direct transmission* occurs through contact with blood and potentially with other body fluids that are contaminated with blood. (**Borne** means carried by. **Contaminated** means not sterile and potentially capable of spreading disease.)

During dental treatment, saliva is *always* considered to be contaminated with blood. This is true even when blood is not

visible in the saliva. Because dental treatment often involves contact with blood and sa-

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Table 5-1 • POTENTIAL DISEASE TRANSMISSION IN THE DENTAL OFFICE

Transmission Route	Transmission Mode
Staff to patient	Infection from a staff member's nose, mouth, or hands may be spread to the patient via airborne transmission or by cross-contamination during treatment.
Patient to staff	Infectious organisms, either airborne or bloodborne, sprayed from the patient's mouth can be transmitted to the dentist, hygienist, or assistant through his or her own nose or mouth or through a break in the skin.
Patient to patient	Transmission occurs through cross-contamination. This may happen when instruments or materials contaminated during the treatment of one patient are not sterilized or are not disinfected properly before use in the treatment of another patient.

liva, bloodborne diseases are of major concern in the dental office.

Splash. Bloodborne diseases may be transmitted during a dental procedure by splashing the mucosa or nonintact skin with blood or blood-contaminated saliva.

Parenteral Transmission. Parenteral transmission can occur when bloodborne pathogens pass through the skin by means of needlestick injuries, human bites, cuts, abrasions, or any other

break in the skin. (The term **parenteral** means through the skin.)

Airborne Transmission

Airborne transmission, also known as *droplet infection*, is the spread of disease by droplets of moisture containing bacteria or viruses. These droplets are spread as people cough, talk, breathe, sing, or sneeze. (When someone sneezes, he sprays contaminated particles out about 8 feet and up about 4 feet.) Diseases such as tuberculosis, measles, and the common cold are transmitted in this manner. The portals of entry for airborne transmission are the nose and mouth.

Aerosol Spray. The high-speed handpiece and ultrasonic scaler create an aerosol spray that is an airborne infection hazard for the dentist, hygienist, and assistant (Figure omitted). (The use of these instruments is discussed in Chapter 18.)

The primary concern with aerosol sprays is the hazard of inhaling them. Inhaling the bacteria and debris in this aerosol spray is approximately the equivalent of having someone sneeze in your face twice per minute—at a distance of 1 foot!

To minimize the risk when treating a patient with a disease that is spread by airborne transmission, the use of the ultrasonic scalers and high-speed handpieces is contraindicated or severely limited. Dental dam is used to isolate the area, and the high-volume oral evacuation (HVE) system is used to reduce the flow of airborne particles. (The use of HVE is discussed in Chapter 16. Dental dam is discussed in Chapter 17.)

Mists. Mists are droplet particles larger than those generated by the aerosol spray. Mists, such as those from coughing, can transmit respiratory infections; however, they are not known to transmit HBV or HIV despite being inhaled.

Spatter. Spatter consists of large droplet particles contaminated with blood, saliva, and other debris. Spatter is created during all restorative and hygiene procedures involving the use of rotary and ultrasonic dental instruments. It may be also created by the use of the air-water syringe.

Spatter droplets travel farther than aerosol mist and tend to land on the upper surfaces of the wrist and forearms, upper arms, and chest and may reach the necktie-collar area of the dentist, assistant, and/or hygienist. HIV, tuberculosis, hepatitis, the herpes-virus, and respiratory illnesses all may be transmitted in this manner.

The appropriate use of dental dam, the HVE system, and proper patient positioning all help to minimize airborne disease transmission during patient treatment.

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[Add. 195]

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MAINE

SIDNEY ABBOTT, Plaintiff	]]	
v.	]	Civil Action No. 94-0273-B
RANDON BRAGDON, D.M.D., Defendant	]]	

Declaration of Deborah Greenspan,
BDS, DSc, ScD(hc), FDS RCSEd (Hon)

Pursuant to 28 U.S.C. § 1746, I Deborah Greenspan, BDS, DSc, ScD (hc), FDS RCSEd (hon) declare that:

I hold a Bachelor of Dental Surgery and a Doctor of Science in oral medicine from the University of London, England, an honorary Doctor of Science from Georgetown University, Washington, D.C., and an honorary Fellowship in Dental Surgery from the Royal College of Surgeons at Edinburgh. I am currently Clinical Professor of Oral Medicine at the University of California at San Francisco and am also Chair of the Committee on Infection Control and Compliance at the UCSF School of Dentistry. At the dental school, I have treated over one thousand patients who are HIV-positive. In addition, I have supervised many dental students and residents, who routinely treat HIV-positive patients in the dental clinic and use the same infection control procedures as can and should be used in a private dental office.

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I have written and lectured extensively on HIV and the practice of dentistry. My education, professional experience, and publications are more fully set forth on my Curriculum Vitae, attached hereto.

I have read the following documents generated in this case:

A. Expert Report of Randon Bragdon, DMD

B. Infectious Disease Policy of Randon Bragdon, DMD
C. Expert Report of Sanford Kuvin, MS, MD, DTM & H
D. Expert Report of Deborah Greenspan, BDS, DSc
E. Expert Report of John Molinari, PhD
F. Expert Report of Donald Craven, MD
G. Deposition of Randon Bragdon, DMD

In addition, I have read the deposition of Randon Bragdon, D.M.D., as well as the exhibits attached thereto, in the case known as John Doe v. Bragdon, C.A. No. CV-93-63 (Me. Sup. Ct.).

Based on my education, experience and expertise I offer the following opinions in this case:

. . . .

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17. In 1988, the American Dental Association's House of Delegates adopted its current policy on the treatment of persons with HIV or AIDS. The policy states, among other things:

> The Association believes that HIV-infected individuals should be treated with compassion and dignity. Current scientific and epidemiologic evidence indicates that there is little risk of transmission of infectious diseases through dental treatment if recommended infection control procedures are routinely followed. Patients with HIV infection may be safely treated in private dental offices when appropriate infection control procedures are employed. Such infection control procedures provide protection both for patients and dental personnel.
>
> A sound approach to the treatment of infectious patients requires an assessment of the patient's condition based on reasonable and informed medical judgments, given the state of medical knowledge at the time. A dentist should

not refuse to treat a patient whose condition is within the dentist's current realm of competence solely because the patient is HIV-infected.

American Dental Association, 132nd Annual Session 539 (1991).

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CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service

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Recommended Infection-Control Practices for Dentistry, 1993

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Recommended Infection-Control Practices for Dentistry, 1993

Summary

This document updates previously published CDC recommendations for infection-control practices in dentistry to reflect new data, materials, technology, and equipment. When implemented, these recommendations should reduce the risk of disease transmission in the dental environment, from patient to dental health-care worker (DHCW), from DHCW to patient, and from patient to patient. Based on principles of infection control, the document delineates specific recommendations related to vaccination of DHCWs; protective attire and barrier techniques; handwashing and care of hands; the use and care of sharp instruments and needles; sterilization or disinfection of instruments; cleaning and disinfection of the dental unit and environmental surfaces; disinfection and the dental laboratory; use and care of handpieces, antiretraction valves, and other intraoral dental devices attached to air and water lines of dental units; single-use disposable instruments; the handling of biopsy specimens; use of extracted teeth in dental educational settings; disposal of waste materials; and implementation of recommendations.

INTRODUCTION

This document updates previously published CDC recommendations for infection-control practices for dentistry (1-3) and offers guidance for reducing the risks of disease transmission among dental health-care workers (DHCWs) and their patients. Although the principles of infection control remain unchanged, new technologies, materials, equipment, and data require continuous evaluation of current infection-control practices. The unique nature of most dental procedures, instrumentation, and patient-care settings also may require specific strategies directed to the prevention of transmission of pathogens among DHCWs and their

patients. Recommended infection-control practices are applicable to all settings in which dental treatment is provided. These recommended practices should be observed in addition to the practices and procedures for worker protection required by the Occupational Safety and Health Administration (OSHA) final rule on Occupational Exposure to Bloodborne Pathogens (29 CFR 1910, 1030), which was published in the *Federal Register* on December 6, 1991 (*4*).

Dental patients and DHCWs may be exposed to a variety of microorganisms via blood or oral or respiratory secretions. These microorganisms may include cytomegalovirus, hepatitis B virus (HBV), herpes simplex virus types 1 and 2, human immunodeficiency virus (HIV), *Mycobacterium tuberculosis*, staphylococci, streptococci, and other viruses and bacteria -- specifically, those that infect the upper respiratory tract. Infections may be transmitted in the dental operatory through several routes, including direct contact with blood, oral fluids, or other secretions; indirect contact with contaminated instruments, operatory equipment, or environmental surfaces; or contact with airborne contaminants present in either droplet spatter or aerosols of oral and respiratory fluids. Infection via any of these routes requires that all three of the following conditions be present (commonly referred to as "the chain of infection"): a susceptible host; a pathogen with sufficient infectivity and numbers to cause infection; and a portal through which the pathogen may enter the

host. Effective infection-control strategies are intended to break one or more of these "links" in the chain, thereby preventing infection.

A set of infection-control strategies common to all health-care delivery settings should reduce the risk of transmission of infectious diseases caused by bloodborne pathogens such as HBV and HIV (*2, 5-10*). Because all infected patients cannot be identified by medical history, physical examination, or laboratory

tests, CDC recommends that blood and body fluid precautions be used consistently for all patients (*2,5*). This extension of blood and body fluid precautions, referred to as "universal precautions," must be observed routinely in the care of all dental patients (*2*). In addition, specific actions have been recommended to reduce the risk of tuberculosis transmission in dental and other ambulatory health-care facilities (*11*).

CONFIRMED TRANSMISSION OF HBV AND HIV IN DENTISTRY

Although the possibility of transmission of bloodborne infections from DHCWs to patients is considered to be small (*12-15*), precise risks have not been quantified in the dental setting by carefully designed epidemiologic studies. Reports published from 1970 through 1987 indicate nine clusters in which patients were infected with HBV associated with treatment by an infected DHCW (*16-25*). In addition, transmission of HIV to six patients of a dentist with acquired immunodeficiency syndrome has been reported (*26, 27*). Transmission of HBV from dentists to patients has not been reported since 1987, possibly reflecting such factors as incomplete ascertainment and reporting, increased adherence to universal precautions -- including routine glove use by dentists -- and increased levels of immunity due to use of hepatitis B vaccine. However, isolated sporadic cases of infection are more difficult to link with a health-care worker than are outbreaks involving multiple patients. For both HBV and HIV, the precise event or events resulting in transmission of infection in the dental setting have not been determined; epidemiologic and laboratory data indicate that these infections probably were transmitted from the DHCWs to patients, rather than from one patient to another (*26,28*). Patient-to-patient transmission of bloodborne pathogens has been reported, however, in several medical settings (*29-31*).

VACCINES FOR DENTAL HEALTH-CARE WORKERS

Although HBV infection is uncommon among adults in the United States (1%-2%), serologic surveys have indicated that 10%-30% of health-care or dental workers show evidence of past or

present HBV infection (*6,32*). The OSHA bloodborne pathogens final rule requires that employers make hepatitis B vaccinations available without cost to their employees who may be exposed to blood or other infectious materials (*4*). In addition, CDC recommends that all workers, including DHCWs, who might be exposed to blood or blood-contaminated substances in an occupational setting be vaccinated for HBV (*6-8*). DHCWs also are at risk for exposure to and possible transmission of other vaccine-preventable diseases (*33*); accordingly, vaccination against influenza, measles, mumps, rubella, and tetanus may be appropriate for DHCWs.

PROTECTIVE ATTIRE AND BARRIER TECHNIQUES

For protection of personnel and patients in dental-care settings, medical gloves (latex or vinyl) always must be worn by DHCWs when there is potential for contacting blood, blood-contaminated saliva, or mucous membranes (*1,2,4-6*). Nonsterile

gloves are appropriate for examination and other nonsurgical procedures (*5*); sterile gloves should be used for surgical procedures. Before treatment of each patient, DHCWs should wash their hands and put on new gloves; after treatment of each patient or before leaving the dental operatory, DHCWs should remove and discard gloves, then wash their hands. DHCWs always should wash their hands and reglove between patients. Surgical or examination gloves should not be washed before use; nor should they be washed, disinfected, or sterilized for reuse. Washing of gloves may cause "wicking" (penetration of liquids through undetected holes in the gloves) and is not recommended (*5*). Deterioration of gloves may be caused by disinfecting agents, oils, certain oil-based lotions, and heat treatments, such as autoclaving.

Chin-length plastic face shields or surgical masks and protective eyewear should be worn when splashing or spattering of

blood or other body fluids is likely, as is common in dentistry (*2,5,6,34,35*). When a mask is used, it should be changed between patients or during patient treatment if it becomes wet or moist. Face shields or protective eyewear should be washed with an appropriate cleaning agent and, when visibly soiled, disinfected between patients.

Protective clothing such as reusable or disposable gowns, laboratory coats, or uniforms should be worn when clothing is likely to be soiled with blood or other body fluids (*2,5,6*). Reusable protective clothing should be washed, using a normal laundry cycle, according to the instructions of detergent and machine manufacturers. Protective clothing should be changed at least daily or as soon as it becomes visibly soiled (*9*). Protective garments and devices (including gloves, masks, and eye and face protection) should be removed before personnel exit areas of the dental office used for laboratory or patient-care activities.

Impervious-backed paper, aluminum foil, or plastic covers should be used to protect items and surfaces (e.g., light handles or x-ray unit heads) that may become contaminated by blood or saliva during use and that are difficult or impossible to clean and disinfect. Between patients, the coverings should be removed (while DHCWs are gloved), discarded, and replaced (after ungloving and washing of hands) with clean material.

Appropriate use of rubber dams, high-velocity air evacuation, and proper patient positioning should minimize the formation of droplets, spatter, and aerosols during patient treatment. In addition, splash shields should be used in the dental laboratory.

HANDWASHING AND CARE OF HANDS

DHCWs should wash their hands before and after treating each patient (i.e., before glove placement and after glove removal) and after barehanded touching of inanimate objects likely to be contaminated by blood, saliva, or respiratory secretions (*2,5,6,9*). Hands should be washed after removal of gloves because gloves may become perforated during use, and DHCWs' hands may become contaminated through contact with patient material. Soap

and water will remove transient microorganisms acquired directly or indirectly from patient contact (*9*); therefore, for many routine dental procedures, such as examinations and nonsurgical techniques, handwashing with plain soap is adequate. For surgical procedures, an antimicrobial surgical handscrub should be used (*10*).

When gloves are torn, cut, or punctured, they should be removed as soon as patient safety permits. DHCWs then should wash their hands thoroughly and reglove to complete the dental procedure. DHCWs then should wash their hands thoroughly and reglove to complete the dental procedure. DHCWs who have exudative lesions or weeping dermatitis, particularly on the hands, should refrain from all direct patient care and from handling dental patient-care equipment until the condition resolves (*12*).
Guidelines

addressing management of occupational exposures to blood and other fluids to which universal precautions apply have been published previously (*6-8,36*).

USE AND CARE OF SHARP INSTRUMENTS AND NEEDLES

Sharp items (e.g., needles, scalpel blades, wires) contaminated with patient blood and saliva should be considered as potentially infective and handled with care to prevent injuries (*2,5,6*).

Used needles should never be recapped or otherwise manipulated utilizing both hands, or any other technique that involves directing the point of a needle toward any part of the body (*2,5,6*). Either a one-handed "scoop" technique or a mechanical device designed for holding the needle sheath should be employed. Used disposable syringes and needles, scalpel blades, and other

sharp items should be placed in appropriate puncture-resistant containers located as close as is practical to the area in which the items were used (*2,5,6*). Bending or breaking of needles before disposal requires unnecessary manipulation and thus is not recommended.

Before attempting to remove needles from nondisposable aspirating syringes, DHCWs should recap them to prevent injuries. Either of the two acceptable techniques may be used. For procedures involving multiple injections with a single needle, the unsheathed needle should be placed in a location where it will not become contaminated or contribute to unintentional needlesticks between injections. If the decision is made to recap a needle between injections, a one-handed "scoop" technique or a mechanical device designed to hold the needle sheath is recommended.

STERILIZATION OR DISINFECTION OF INSTRUMENTS

Indications for Sterilization or Disinfection of Dental Instruments

As with other medical and surgical instruments, dental instruments are classified into three categories -- critical, semicritical, or noncritical -- depending on their risk of transmitting infection and the need to sterilize them between uses (*9,37-40*). Each dental practice should classify all instruments as follows:

- **Critical**. Surgical and other instruments used to *penetrate soft tissue or bone* are classified as critical and should be sterilized after each use. These devices include forceps, scalpels, bone chisels, scalers, and burs.

- **Semicritical.** Instruments such as mirrors and amalgam condensers that *do not penetrate soft tissues or bone but contact oral tissues* are classified as semicritical. These devices should be sterilized after each use. If, however, sterilization is not feasible because the instrument will be damaged by heat, the

instrument should receive, at a minimum, high-level disinfection.

- **Noncritical.** Instruments or medical devices such as external components of x-ray heads that *come into contact only with intact skin* are classified as noncritical. Because these noncritical surfaces have a relatively low risk of transmitting infection, they may be reprocessed between patients with intermediate-level or low-level disinfection (see Cleaning and Disinfection of Dental Unit and Environmental Surfaces) or detergent and water washing, depending on the nature of the surface and the degree and nature of the contamination (*9,38*).

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Methods of Sterilization or Disinfection of Dental Instruments

Before sterilization or high-level disinfection, instruments should be cleaned thoroughly to remove debris. Persons involved in cleaning and reprocessing instruments should wear heavy-duty (reusable utility) gloves to lessen the risk of hand injuries. Placing instruments into a container of water or disinfectant/detergent as soon as possible after use will prevent drying of patient material and make cleaning easier and more efficient. Cleaning may be accomplished by thorough scrubbing with soap and water or a detergent solution, or with a mechanical device (e.g., an ultrasonic cleaner). The use of covered ultrasonic cleaners, when possible, is recommended to increase efficiency of cleaning and to reduce handling of sharp instruments.

All critical and semicritical dental instruments that are heat stable should be sterilized routinely between uses by steam under pressure (autoclaving), dry heat, or chemical vapor, following the instructions of the manufacturers of the instruments and the sterilizers. Critical and semicritical instruments that will not be used immediately should be packaged before sterilization.

Proper functioning of sterilization cycles should be verified by the periodic use (at least weekly) of biologic indicators (i.e, spore tests) (*3,9*). Heat-sensitive chemical indicators (e.g., those that change color after exposure to heat) alone do not ensure adequacy of a sterilization cycle but may be used on the outside of each pack to identify packs that have been processed through the heating cycle. A simple and inexpensive method to confirm heat penetration to all instruments during each cycle is the use of a chemical indicator inside and in the center of either a load of unwrapped instruments or in each multiple instrument pack (*41*); this procedure is recommended for use in all dental practices. Instructions provided by the manufacturer of medical/dental instruments and sterilization devices should be followed closely.

In all dental and other health-care settings, indications for the use of liquid chemical germicides to sterilize instruments (i.e., "cold sterilization") are limited. For heat-sensitive instruments, this procedure may require up to 10 hours of exposure to a liquid chemical agent, registered with the U.S. Environmental Protection Agency (EPA) as a "sterilant/disinfectant." This sterilization process should be followed by aseptic rinsing with sterile water, drying, and, if the instrument is not used immediately, placement in a sterile container.

EPA-registered "sterilant/disinfectant" chemicals are used to attain high-level disinfection of heat-sensitive semicritical medical and dental instruments. The product manufacturers' directions regarding appropriate concentration and exposure time should be followed closely. The EPA classification of the liquid chemical agent (i.e., "sterilant/disinfectant") will be shown on the chemical label. Liquid chemical agents that are less potent than the "sterilant/disinfectant" category are *not* appropriate for reprocessing critical or semicritical dental instruments.

CLEANING AND DISINFECTION OF DENTAL UNIT AND ENVIRONMENTAL SURFACES

After treatment of each patient and at the completion of daily work activities, countertops and dental unit surfaces that may

have become contaminated with patient material should be cleaned with disposable toweling, using an appropriate cleaning agent and water as necessary. Surfaces then should be disinfected with a suitable chemical germicide.

A chemical germicide registered with the EPA as a "hospital disinfectant" and labeled for "tuberculocidal" (i.e., mycobactericidal) activity is recommended for

disinfecting surfaces that have been soiled with patient material. These intermediate-level disinfectants include phenolics, iodophors, and chlorine-containing compounds. Because mycobacteria are among the most resistant groups of microorganisms, germicides effective against mycobacteria should be effective against many other bacterial and viral pathogens (*9,38-40,42*). A fresh solution of sodium hypochlorite (household bleach) prepared daily is an inexpensive and effective intermediate-level germicide. Concentrations ranging from 500 to 800 ppm of chlorine (a 1:100 dilution of bleach and tap water or ¼ cup of bleach to 1 gallon of water) are effective on environmental surfaces that have been cleaned of visible contamination. Caution should be exercised, since chlorine solutions are corrosive to metals, especially aluminum.

Low-level disinfectants -- EPA-registered "hospital disinfectants" that are not labeled for "tuberculocidal" activity (e.g., quaternary ammonium compounds) -- are appropriate for general housekeeping purposes such as cleaning floors, walls, and other housekeeping surfaces. Intermediate- and low-level disinfectants are *not* recommended for reprocessing critical or semicritical dental instruments.

DISINFECTION AND THE DENTAL LABORATORY

Laboratory materials and other items that have been used in the mouth (e.g., impressions, bite registrations, fixed and removable prostheses, orthodontic appliances) should be cleaned and disinfected before being manipulated in the laboratory, whether an on-site or remote location (*43*). These items also should be cleaned and disinfected after being manipulated in the dental laboratory and before placement in the patient's mouth (*2*). Because of the increasing variety of dental materials used intraorally, DHCWs are advised to consult with manufacturers regarding the stability of specific materials relative to disinfection procedures. A chemical germicide having at least an intermediate level of activity (i.e., "tuberculocidal hospital disinfectant") is appropriate for such disinfection. Communication between dental office and dental laboratory personnel regarding the handling and decontamination of supplies and materials is important.

USE AND CARE OF HANDPIECES, ANTIRETRACTION VALVES, AND OTHER INTRAORAL DENTAL DEVICES ATTACHED TO AIR AND WATER LINES OF DENTAL UNITS

Routine between-patient use of a heating process capable of sterilization (i.e., steam under pressure (autoclaving), dry heat, or heat/chemical vapor) is recommended for all high-speed dental handpieces, low-speed handpiece components used introrally and reusable prophylaxis angles. Manufacturers' instructions for cleaning, lubrication, and sterilization procedures should be followed closely to ensure both the effectiveness of the sterilization process and the longevity of these instruments. According to manufacturers, virtually all high-speed and low-speed handpieces in production today are heat tolerant, and most heat-sensitive models manufactured earlier can be retrofitted with heat-stable components.

Internal surfaces of high-speed handpieces, low-speed handpiece components, and prophylaxis angles may become contaminated with patient material during use. This retained

patient material then may be expelled intraorally during subsequent uses (*44-46*). Restricted physical access -- particularly to internal surfaces of these instruments -- limits cleaning and disinfection or sterilization with liquid chemical

germicides. Surface disinfection by wiping or soaking in liquid chemical germicides is *not* n acceptable method for reprocessing high-speed handpieces, low-speed handpiece components used intraorally, or reusable prophylaxis angles.

Because retroaction valves in dental unit water lines may cause aspiration of patient material back into the handpiece and water lines, antiretraction valves (one-way flow check valves) should be installed to prevent fluid aspiration and to reduce the risk of transfer of potentially infective material (*47*). Routine maintenance of antiretraction valves is necessary to ensure effectiveness; the dental unit manufacturer should be consulted to establish an appropriate maintenance routine.

High-speed handpieces should be run to discharge water and air for a minimum of 20-30 seconds after use on each patient. This procedure is intended to aid in physically flushing out patient material that may have entered the turbine and air or water lines (*46*). Use of an enclosed container or high-velocity evacuation should be considered to minimize the spread of spray, spatter, and aerosois generated during discharge procedures. Additionally, there is evidence that overnight or weekend microbial accumulation in water lines can be reduced substantially by removing the handpiece and allowing water lines to run and to discharge water for several minutes at the beginning of each clinic day (*48*). Sterile saline or sterile water should be used as a coolant/irrigator when surgical procedures involving the cutting of bone are performed.

Other reusable intraoral instruments attached to, but removable from, the dental unit air or water lines -- such as ultrasonic scaler tips and component parts and air/water syringe tips

-- should be cleaned and sterilized after treatment of each patient in the same manner as handpieces, which was described previously. Manufacturers' directions for reprocessing should be followed to ensure effectiveness of the process as well as longevity of the instruments.

Some dental instruments have components that are heat sensitive or are permanently attached to dental unit water lines. Some items may not enter the patient's oral cavity, but are likely to become contaminated with oral fluids during treatment procedures, including, for example, handles or dental unit attachments of saliva ejectors, high-speed air evacuators, and air/water syringes. These components should be covered with impervious barriers that are changed after each use or, if the surface permits, carefully cleaned and then treated with a chemical germicide having at least an intermediate level of activity. As with high-speed dental handpieces, water lines to all instruments should be flushed thoroughly after the treatment of each patient; flushing at the beginning of each clinic day also is recommended.

SINGLE-USE DISPOSABLE INSTRUMENTS

Single-use disposable instruments (e.g., prophylaxis angles; prophylaxis cups and brushes, tips for high-speed air evacuators, saliva ejectors, and air/water syringes) should be used for one patient only and discarded appropriately. These items are neither designed nor intended to be cleaned, disinfected, or sterilized for reuse.

HANDLING OF BIOPSY SPECIMENS

In general, each biopsy specimen should be put in a sturdy container with a secure lid to prevent leaking during transport. Care should be taken when collecting specimens to avoid contamination of the outside of the container. If the outside of the container is visibly contaminated, it should be cleaned and disinfected or placed in an impervious bag (*49*).

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USE OF EXTRACTED TEETH IN DENTAL EDUCATIONAL SETTINGS

Extracted teeth used for the education of DHCWs should be considered infective and classified as clinical specimens because they contain blood. All persons who collect, transport, or manipulate extracted teeth should handle them with the same precautions as a specimen for biopsy (*2*). Universal precautions should be adhered to whenever extracted teeth are handled; because preclinical educational exercises simulate clinical experiences, students enrolled in dental educational programs should adhere to universal precautions in both preclinical and clinical settings. In addition, all persons who handle extracted teeth in dental educational settings should receive hepatitis B vaccine (*6-8*).

Before extracted teeth are manipulated in dental educational exercises, the teeth first should be cleaned of adherent patient material by scrubbing with detergent and water or by using an ultrasonic cleaner. Teeth should then be stored, immersed in a fresh solution of sodium hypochlorite (household bleach diluted 1:10 with tap water) or any liquid chemical germicide suitable for clinical specimen fixation (*50*).

Persons handling extracted teeth should wear gloves. Gloves should be disposed of properly and hands washed after completion of work activities. Additional personal protective equipment (e.g., face shield or surgical mask and protective eyewear) should be worn if mucous membrane contact with debris or spatter is anticipated when the specimen is handled, cleaned, or manipulated. Work surfaces and equipment should be cleaned and decontaminated with an appropriate liquid chemical germicide after completion of work activities (*37,38,40,51*).

The handling of extracted teeth used in dental educational settings differs from giving patients their own extracted teeth. Several states allow patients to keep such teeth, because these teeth

are not considered to be regulated (patholigic) waste (*52*) or because the removed body part (tooth) becomes the property of the patient and does not enter the waste system (*53*).

DISPOSAL OF WASTE MATERIALS

Blood, suctioned fluids, or other liquid waste may be poured carefully into a drain connected to a sanitary sewer system. Disposable needles, scalpels, or other sharp items should be placed intact into puncture-resistant containers before disposal. Solid waste contaminated with blood or other body fluids should be placed in sealed, sturdy impervious bags to prevent leakage of the contained items. All contained solid waste should then be disposed of according to requirements established by local, state, or federal environmental regulatory agencies and published recommendations (*9,49*).

IMPLEMENTATION OF RECOMMENDED INFECTION-CONTROL PRACTICES FOR DENTISTRY

Emphasis should be placed on consistent adherence to recommended infection-control strategies, including the use of protective barriers and appropriate methods of sterilizing or disinfecting instruments and environmental surfaces. Each dental facility should develop a written protocol for instrument reprocessing, operatory cleanup, and management of injuries (*3*). Training of all DHCWs in proper infection-control practices should begin in professional and vocational schools and be updated with continuing education.

ADDITIONAL NEEDS IN DENTISTRY

Additional information is needed for accurate assessment of factors that may increase the risk for transmission of bloodborne

pathogens and other infectious agents in a dental setting. Studies should address the nature, frequency, and circumstances of occupational exposures. Such information may lead to the development and evaluation of improved designs for dental instruments, equipment and personal protective devices. In addition, more efficient reprocessing techniques should be considered in the design of future dental instruments and equipment. Efforts to protect both patients and DHCWs should include improved surveillance, risk assessment, evaluation of measures to prevent exposure, and studies of postexposure prophylaxis. Such efforts may lead to development of safer and more effective medical devices, work practices, and personal protective equipment that are acceptable to DHCWs, are practical and economical, and do not adversely affect patient care (*54,55*).

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HIV TRANSMISSION IN THE HEALTH-CARE SETTING

Risks to Health-Care Workers and Patients

Laurie M. Robert, MS, and David M. Bell, MD

Evaluating the risk of HIV transmission in the health-care setting requires data from various sources, including surveillance activities that attempt to ascertain cases of HIV transmission to health-care workers (HCWs) and patients, seroprevalence studies among HCWs and patients, and prospective studies that evaluate a HCW's risk of seroconverting following an exposure to HIV-infected blood and that attempt to define the frequency and preventability of blood contacts in health-care settings.

TRANSMISSION FROM PATIENT TO HEALTH-CARE WORKER

Surveillance Data

Information on employment in a health-care setting is collected on AIDS case reports that are sent by state or local health departments to the Centers for Disease Control and Prevention (CDC). As of March 31, 1993, this information was available for 214,686 adults with AIDS; 10,122 (4.7%) had been employed in a health-care setting. In comparison, approximately 5.7% of the United States labor force is employed in health services.[7] Of the HCWs with AIDS, 94% reported nonoccupational risks for acquiring HIV. The remaining 6% are reported as having an undetermined risk; most of these are still under investigation to determine the mode of transmission (C. Ciesielski, personal communication, 1993). HCWs who do not have another risk identified after follow-up investigation and who state they had previous occupational exposure to blood or body fluids are classified as cases of possible occupational transmission.

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As of June 30, 1993, a total of 115 US workers have been reported to a national surveillance system for occupational transmission of HIV (Table 1). Thirty-seven workers had documented seroconversion associated with a documented exposure to HIV. Most of the exposures[34] were to HIV-infected blood, one to visibly bloody fluid, one to an unspecified fluid, and one to concentrated virus in a laboratory. Thirty-two of the 37 workers had percutaneous exposures, four had mucocutaneous, and one had both percutaneous and mucocutaneous exposures. Eight of these HCWs have developed AIDS. Another 78 HCWs have been reported as possible cases of occupational acquisition of HIV. These workers did not report non-occupational risks for HIV and have reported occupational exposures to blood, body fluids, or laboratory solutions containing HIV; however, seroconversion was not documented, usually because no baseline sera were available to establish seronegativity at the time of exposure.[8]

This passive surveillance system for occupational transmission most likely does not reflect the full extent of occupational transmission; such cases can be difficult to document, particularly if exposures are not reported at the time of occurrence. In the past, it has been estimated that only 10% to 60% of percutaneous injuries are reported.[28,36]

HIV Seroprevalence Among Health-Care Workers

HIV seroprevalence surveys provide a means of indirectly assessing HCWs' risk of HIV infection (Table 2). CDC has sponsored two voluntary anonymous seroprevalence surveys of surgeons. In 1992, a serosurvey was performed among surgeons in three specialties practicing in moderate to high AIDS incidence areas.

Table 1. US HEALTH-CARE WORKERS WITH DOCUMENTED AND POSSIBLE OCCUPATIONALLY ACQUIRED HIV INFECTION, REPORTED THROUGH JUNE 1993

Occupation	No. of Cases of Documented Occupational Transmission	No. of Cases of Possible Occupational Transmission
Dental worker, including dentist	-	7
Embalmer/morgue technician	-	3
Emergency medical technician/paramedic	-	7
Health aide/attendant	1	8
Housekeeper/maintenance worker	1	6
Laboratory technician, clinical	14	13
Laboratory technician, nonclinical	1	1
Nurse	13	15
Physician, nonsurgical	4	8
Physician, surgical	-	2
Respiratory therapist	1	1
Technician, dialysis	1	1
Technician, surgical	1	1
Technician/therapist, other than those listed above	-	3
Other health-care occupations	-	2
Total	37	78

Health-care workers are defined as those persons, including students and trainees, who have worked in a health-care, clinical, or HIV laboratory setting at any time since 1978. (*From* Centers for Disease Control HIV/AIDS Surveillance Report, July 1993.)

Table 2. PUBLISHED HIV SEROPREVALENCE IN SELECTED HEALTH-CARE WORKERS

Occupation Author	No. Tested	No. Positive	Percent Prevalence*
Surgeons			
Panlilio et al[40]	770	1	0.14
Tokars et al[52]	3420	2	0
Physicians and dentists (US Army Reserve) Cowan et al[16]	3347	3	Not available
Dentists			
Gerberding et al[22]	304†	0	0
Flynn et al[20]	89	0	0
Klein et al[34]	1132†	1	0.9
Siew et al[50]	1195	0	0
Gruninger et al[26]	1480	0	0
Ebbesen et al[10]	961	0	0
Dental hygienists			
Flynn et al[20]; Klein et al[34]	167	0	0
Dental assistants	176	0	0
Flynn et al[20]; Klein et al[34]			
Hemodialysis staff	356	0	0
Assogba et al[3]; Chirgwin et al[12]; Comodo et al[16];			
Health-care worker blood donors			
Chamberland et al[10]	9449	3	‡

*Excluding seropositives with nonoccupational risk.
†Persons with community risk excluded.
‡One HCW lost to follow-up

Among the 770 participants, one surgeon, who did not report nonoccupational risks for HIV infection on an anonymous questionnaire, was HIV seropositive. This surgeon practiced general surgery for over 25 years and reported three percutaneous injuries involving patients with HIV infection or AIDS since 1978.[40] At the annual meting of the American Academy of Orthopedic Surgeons in 1991, two of 3420 surgeons who participated in the survey were HIV seropositive; both reported nonoccupational risk factors.[52]

In a testing program conducted among physicians, surgeons, and dentists in the US Army Reserve, who normally spend 90% of their time in civilian life, the HIV seroprevalence was 0 of 263 among women and 3 of 3084 among men. No other information regarding possible risk factors was reported for these seropositive men.[16]

No seropositives were found among 356 hemodialysis staff in five cities.[3,12,15,23,43] Three (0.03%) of 9449 HCW blood donors evaluated in six urban regions between March 1990 and May 1991 were HIV seropositive; two reported nonoccupational risk factors for HIV, and one was lost to follow-up.[10] Studies of dentists, dental hygienists, and dental chairside assistants have found low rates of infection among persons not reporting behavioral or transfusion risks (Table 2).[19,20,22,26,34,50]

A major limitation of HIV seroprevalence studies in HCWs to date is that the extent of occupational or nonoccupational exposure of most workers tested is not known. Also, some of these rates may be underestimates if workers who knew or suspected that they might be positive declined to be tested. Although these rates may not be generalized to all HCWs, these seroprevalence surveys indicate that

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there is not a high rate of previously undetected HIV infection in the HCWs studied, many of whom had substantial opportunity for occupational exposures.

RISK OF TRANSMISSION

Estimates of the occupational risk of HIV infection can be derived from data on the nature and frequency of HCW exposure to blood or bloody body fluids, HIV seroprevalence of patients, and the risk of infection transmission after a single exposure.

Epidemiology of Blood Contact

Understanding of the nature, frequency, and preventability of blood contacts (i.e., percutaneous injuries and mucocutaneous contacts) among HCWs and patients has been advanced by prospective studies in surgery and obstetrics (Table 3) in emergency departments and on hospital wards.[27,32,35,37,47,55]

For example, in 1990, Tokars et al[51] observed 1382 operations in five surgical specialties (general, orthopedic, gynecology, trauma, and cardiac) in two urban and two suburban US hospitals in areas of high AIDS incidence. In 6.9% of procedures, a total of 99 percutaneous injuries was observed; most were related to suturing. Rates were highest on the gynecology service (10%).In a logistic regression model, independent risk factors for injury included the type of procedure performed, its duration, and holding tissue being sutured with fingers rather than an instrument.

Preliminary data from a study in 1992 to 1993 in three hospitals in a high

Table 3. OBSERVATIONAL STUDIES OF BLOOD CONTACTS SUSTAINED IN SURGICAL AND OBSTETRIC PROCEDURES

Author/Location	No. Procedures Observed	Procedures with ≥ 1 Blood Contact (%)	Procedures with ≥ 1 Sharps Injury (%)
Obstetrics			
Rudnick* (New York)	845	26.5	1.3
Panlilio et al[42] (Atlanta)	230	32.2	1.7
Surgery			
Tokars et al[51] (New York, Chicago)	1382	46.6	6.9
Panlilio et al[41] (Atlanta)	206	30.1	4.9
Popejoy and Fry[44] (Albuquerque)	684	27.8	3.1
Quebberman et al[46] (Milwaukee)	234	50.4	15.4
Gerberding et al[21] (San Francisco)	1307	6.4	1.3

*CDC, unpublished data. 1993.

AIDS incidence urban area found that one or more obstetric personnel sustained a mucocutaneous blood contact in 27% and a percutaneous injury in 1% of 845 deliveries. Of the mucocutaneous contacts, 49% might have been prevented by the use of commonly available barrier precautions, such as gloves and gowns (J. Rudnick, CDC, unpublished data, 1993). In 281 gynecologic surgical procedures, one or more personnel sustained mucocutaneous blood contact in 37% and a percutaneous injury in 5%. Of the 16 percutaneous injuries, half were inflicted by a co-worker. Double gloving appeared to be beneficial; 25% of single-gloved physicians, medical students, and scrub nurses but only 1% of those double-gloved sustained blood-skin contacts to the hand.[49]

Prospective studies of percutaneous exposures on medical wards in the United States have found rates of 1.8 per year (0.15 per patient-care month) for a physician[55] and 0.98 per year for a nurse (H. Smith, University of Pennsylvania, personal communication, 1993).

Among 326 needlestick injuries reported to the employee health service at a university hospital, Jagger et al[32] found that 17.8% occurred before or during use, 69.6% occurred after use but before disposal, and 13.2% occurred during or after disposal. One third of injuries involved recapping, which was often reportedly done to avoid a competing hazard, such as disassembling a device with an exposed needle or carrying uncapped items to a disposal box. When injury rates were estimated based on number of devices purchased, the highest rates of injury were for items that required disassembly after use. Among 2248 percutaneous injuries reported in CDC's multicenter surveillance project of HCWs exposed to HIV-infected blood, the greatest percentage of injuries (44%) also occurred after use of a device but before disposal; 38% occurred during use of the device, 10% during disposal, and 7% at other or unknown times. (D.M. Cardo, unpublished CDC data, 1993). These data suggest that a large proportion of needlestick injuries related to phlebotomy and intravenous lines could be prevented with new or modified medical devices. Training, changes in work practices, and improved access to needle disposal containers are also important[4,27,55] but have not been uniformly successful in reducing injury rates.[35]

In recent years, many new devices with features intended to reduce the likelihood of percutaneous injury have been marketed. In most cases, insufficient data are available from clinical trials to assess whether these devices are effective in preventing exposures without compromising patient care. Surveys of user satisfaction provide important information, yet differences in user satisfaction between hospitals highlight the need for objective evaluation. Field trials of new devices in clinical settings have posed daunting challenges. Surveillance of injuries among the many different HCWs who may use the device can be difficult, particularly at large medical centers, where hospital employees, students, residents, and attending physicians may be instructed to report injuries to different locations. Such injuries have been notoriously underreported[28,36]; reporting rates may be influenced by various factors, including the

HCW's impression of the severity of the injury, the likelihood that the source patient has HIV infection, the accessibility of the system, and the perceived response of the employer. To evaluate a new device, an appropriate historical or concurrent control group must be selected; large sample sizes may be required to demonstrate reductions in rates of events occurring at low frequency. Personnel must be trained in the use of the new device, which can be difficult given multiple occupations, shifts, and rotations. It may be difficult to measure other important variables, such as compliance in using the device and in activating its safety features; denominators to estimate device-specific injury rates; to identify and adjust for possible confounding variables (e.g., other preventive measures introduced simultaneously); and to assess the impact of the device on patient care.

The New York State Department of Health recently completed a pilot study of

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new devices that generated important insights and identified certain difficulties.[11] For example, reported IV-related injuries declined 75% to 93.8% in three hospitals after introduction of new IV delivery systems. In the fourth hospital, the reported number of such injuries increased; the increase was attributed to failure of staff to properly utilize the safety system. Some injuries occurred due to improper use of a safety device or during activation of a safety feature. For example, in at least two instances, workers sustained needlestick injuries because their hand slipped as they were trying to slide forward a needle guard on a syringe. Passive devices that do not require activation by the user are more likely to reliably protect against injury.[11,32] CDC is currently collaborating with a number of hospitals in conducting enhanced surveillance of occupational exposures in surgery and on hospital wards, particularly to permit evaluation of new preventive measures.

HIV Seroprevalence Among Patients

In the United States, patient HIV seroprevalence varies widely by geographic area and the patient's age, sex, race/ethnicity, presenting clinical conditions, and other factors. Studies have detected HIV seroprevalence in hospital patients to be 0.2% to 14.2%33 and in emergency department patients to be 0.2% to 8.9%.37 Several studies have indicated that the HIV infection status of a majority of HIV-infected patients was unknown to the health-care provider at the time of presentation,[33,37] emphasizing the need to follow recommendations for universal precautions.[9]

Risk of Infection Postexposure

The risk of HIV infection after a documented exposure to HIV-infected blood has been estimated based on prospective studies of over 3000 HCWs after exposure.[29,31,53] Results from these studies have indicated that the risk of HIV transmission after a single percutaneous exposure to HIV-infected blood is approximately 0.3%.[31,53] In one study, a HCW seroconverted after extensive contact of eyes, mouth, and hands and a large amount of blood from an asymptomatic source patient; aggregating this study with others, the risk after a mucous membrane exposure has been estimated at 0.09%.[31] The risk after a skin exposure to HIV-infected blood is believed to be less but has not been precisely quantified because no HCWs enrolled in these prospective studies have seroconverted after an isolated skin exposure.

The risk of HIV transmission after a percutaneous exposure is likely to depend on several factors. The most important of these is probably the viral titer in the blood of the source patient, which can vary by several orders of magnitude.[30] Data from laboratory studies suggest that injuries with hollow-bore needles result in transfer of greater volumes of blood and that glove use can reduce this volume.[56] To better quantify these variables, CDC is currently developing a case-control study to compare HCWs who became infected after exposure with those who did not.

TRANSMISSION FROM HEALTH-CARE WORKER TO PATIENT

Transmission of HIV to a patient during an invasive procedure performed by a dentist with AIDS was reported in 1990.[10] A comprehensive epidemiologic and

laboratory investigation subsequently identified five additional patients (of approximately 110 tested) who were infected while receiving care from the dentist. All six patients had no other risks for acquiring HIV and all had viral strains that closely matched that of the dentist on genetic sequencing analysis. The precise event(s) resulting in transmission of HIV from the dentist to the patients is not known, but epidemiologic and laboratory evidence strongly suggests provider-to-patient rather than patient-to-patient transmission.[13,24] This remains the only recognized instance of HIV transmission from an infected HCW to patients.

Lookback Investigation Data

Several accounts of retrospective investigations of patients of HIV-infected HCWs have been published.[1,2,14,17,18,38,39,45,48,54,57] As of August 31, 1993, CDC was aware of test results for 22,032 patients treated by 63 HIV-infected HCWs (excluding the Florida dental practice). No HIV infections were found among 13,059 patients tested in the practices of 50 HCWs, including 25 dentists and dental students, 13 physicians and medical students, 8 surgeons and obstetricians, and 4 other HCWs. For the remaining 13 HCWs (8 dentists and 5 surgeons or obstetricians), 8973 patients were tested and 112 seropositive patients were identified. Follow-up investigations have been completed for 106 (95%) of these patients. To date, transmission from an HCW has not been documented as a source of infection for any of the patients tested (L. Robert, CDC, unpublished data, 1993).

Although these data are reassuring, they have certain limitations, due in part to the technical difficulties and confidentiality issues involved in identifying and retrieving various records and blood specimens. Test results are available for only

about 20% of patients of these HCWs. It is not known how many of the patients tested underwent invasive procedures and which procedures these were; specific procedure data are available for only 17% of tested patients. In most cases it is not known what stage of infection the HCWs were in when the procedures were performed; i.e., whether they had developed AIDS, in which the titer of HIV in blood is highest and transmission may be more likely.[30] In other words, data from these retrospective studies, although reassuring, may not necessarily apply to every surgeon or to every procedure.

Because of the difficulties in conducting and interpreting studies of HIV seroprevalence in patients of HIV-infected HCWs, modeling techniques have been used to estimate the risk to patients. The average risk of transmission of HIV and hepatitis B from infected surgeons to patients due to a percutaneous injury during invasive procedures has been estimated as 1 in 42,000 to 1 in 420,000 for HIV and 1 in 420 to 1 in 4200 for a hepatitis B e antigen (HBeAg)-positive surgeon.[3] These estimates, based on limited data and not taking cluster transmission into account, represent population averages and may not necessarily apply to a particular procedure performed by a particular surgeon, for which the risk may be higher or lower than the estimated average.[5,38]

Lessons from Investigations of Transmission of Hepatitis B Virus from HCWs to Patients

Transmission patterns of another blood-borne pathogen, hepatitis B virus (HBV), can be useful in understanding risk factors for HIV transmission but do not assist in defining the magnitude of the risk.[6] HBV is much more transmissible and more environmentally stable than HIV. The risk of infection after a percutaneous

exposure is approximately 30% if the exposure is to hepatitis B e antigen positive blood, compared to 0.3% for HIV-infected blood. Since the 1970s there have been over 350 cases of recognized HBV

transmission to patients, mostly in clusters related to 34 infected providers (21 surgeons, 9 dentists, 1 respiratory therapist, and 2 cardiopulmonary bypass pump technicians).[6] All of these HCWs who were tested for hepatitis B e antigen were positive. As is true for the Florida dentist case, the precise events leading to transmission were not determined in these incidents; however, it is believed that percutaneous blood exposure played a major role. In several investigations of patients of infected gynecologists and dentists, the risk of HBV transmission appeared to increase with the invasiveness of the procedure. In all instances of HBV transmission from infected providers to patients, contamination of surgical wounds or traumatized tissue could potentially have occurred from injuries incurred by the HCW that resulted in recontact to the patient or from breaches in standard infection control practices. In some cases where providers resumed practice after counseling to modify techniques, no further transmission occurred. In several other instances, however, transmission continued even after such changes were made.[6]

The concept that certain procedures are more likely than others to be associated with a percutaneous injury to a surgeon and subsequent exposure to the patient's blood is substantiated in the previously mentioned observational studies. These studies have also illustrated the potential for a patient to be exposed to a surgeon's blood[47,51] for example, among the 88 percutaneous injuries observed in surgeons in the multicenter study of Tokars et al,[51] the sharp object recontacted the patient's open wound after injuring the surgeon in 32% of the injuries. The surgeon injury and patient recontact rates varied by service; they were highest on the gynecology service, and within that service, they were highest for vaginal hysterectomy.[51] Although the risk of infection transmission due to a recontact is unknown, surgeon injury rates and patient recontact rates were elevated in the specialties and procedures most often implicated in HBV transmission from surgeons to patients. This study also suggested that technique changes can potentially reduce injury and recontact rates. In addition to procedures performed, holding tissue being sutured with fingers (rather than an instrument) was identified as an independent risk factor for surgeon injury. Most patient recontacts involved continued use of the needle or sharp object that injured the surgeon, suggesting that

many might have been prevented by timely substitution with a sterile instrument.

Since HIV is not transmitted by casual contact or fomites, patient-to-patient transmission of HIV in health-care settings should be prevented. Patient-to-patient transmission has been rarely reported due to reuse of improperly sterilized needles,[29a] inadvertent injection of blood or other material from previous patients during nuclear medicine procedures,[34a,8a] or as the result of a presumed breach in infection control in a surgeon's office.[10a]

FUTURE ASPECTS FOR PREVENTION

The prevention of exposures in the health-care setting is the best way to prevent transmission of blood-borne pathogens from patient to HCW or HCW to patient. Future efforts to prevent exposures will require the development of improved engineering controls (e.g., safer medical devices) and work practices (e.g., technique change; reducing the need to handle sharps) and personal protective equipment (e.g., impervious materials for barrier precautions), as well as training workers in the appropriate use of these measures. Of premier importance is the need to evaluate new interventions to determine their effectiveness, feasibility, user satisfaction, and their impact on patient care.

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HIV, HEALTH CARE WORKERS AND PATIENTS: HOW TO ENSURE SAFETY IN THE DENTAL OFFICE

JOHN A. MOLINARI, PH.D.

ABSTRACT

What can be done to preserve the high standards of patient care, give reasonable assurances to patients and acknowledge the rights of health care workers? Conscientious adherence to infection control is the most responsible option available.

The professional relationship between many dental and medical health care providers and their patients has been modified substantially during the last 10 to 12 years.

Infectious diseases and infection control have dramatically affected this change. The basic reasons did not involve well-known pathogens such as hepatitis B virus or herpes simplex virus, both documented as significant occupational risks for health care workers and, to a lesser extent, for patients exposed to infected health care workers.[1-7]

Despite numerous reports on these and other infectious disease risks, the major catalyst for the change was the discovery and publication of a new form of life-threatening immune deficiency condition for which we did not (and still do not) have a cure--AIDS.[8,9] The door to widespread concern was opened further when what is now termed human immunodeficiency virus was isolated a few years later.[10-12]

Because of their bloodborne transmission potential, many organizations used both HBV and HIV as prototypical target microorganisms in developing infection control guidelines and regulations. These organizations included the Centers for Disease Control and Prevention,[13-16] the American Dental Association[17,18] and the Occupational Safety and Health Administration.[19]

HCW RISKS FROM HIV-INFECTED PATIENTS

Publication of detailed scientific information characterizing the structure, genetics and chemical susceptibility of HIV, its transmission, progression of infection and chemotherapeutic interventions did not provide all the answers to lessen people's fears. Extensive media coverage about HIV and AIDS, and publicity surrounding celebrities who died from complications of AIDS, caused many HCWs to ask a singular question: How much of an occupational risk do we have when treating HIV-infected and AIDS patients?

Characterization of HIV structural antigens, marker antibodies, along with the development of increasingly sensitive serological assays, provided opportunities to investigate suspected cross-infection cases. Despite thousands of reported accidental exposures, 33 occupational HIV transmissions were documented by the CDC as of April 1993, with 69 additional cases still under investigation (Table).[20]

A few important trends become apparent when reviewing these data:

- while not zero, the risk of HIV transmission to HCWs continues to be very small compared to other bloodborne infections such as hepatitis B;
- no documented HIV transmission to dental workers has yet been reported.

A very important portion of the findings indicates that 24 of 33 documented occupational HIV transmissions and 27 of 69 possible occupational HIV transmissions have occurred in either nurses or laboratory technicians. Both groups routinely use hollow needles and collect blood specimens from patients. Blood has been the most common body fluid

involved in occupational HIV transmission to HCWs. Most reported health care injuries involve needlesticks or other sharps accidents.[20]

Recommendations from organizations like CDC incorporated this clinical knowledge into published recommendations. These recommendations emphasize the need for HCWs first to consider all patients as potentially infected with HIV or other bloodborne pathogens. Second, they call for rigorous use of infection control precautions to minimize the risk of exposure to blood and other body fluids of all patients.[21]

With specific reference to HIV risks for dental professionals, six cases have been investigated and are currently without identifiable behavioral or transfusion risks. In each instance, percutaneous or mucocutaneous occupational exposure to blood or other body fluids containing HIV was reported, but HIV seroconversion was not documented. A study carried out by the ADA as part of its annual session Health Screening Program suggested further that dentists are at small risk of occupationally acquired HIV infection.[22]

Though current data suggest a very low HIV risk for dental care providers, these data should not be interpreted as indicating no risk. Our routine aseptic, barrier, disinfection and sterilization infection control protocols have gone a long way in providing high standards of practice safety for both treatment providers and those receiving care. These standards should not be compromised just because HIV does not seem to be a significant occupational pathogen.

PATIENT RISKS FROM HIV-INFECTED HCWS

Studies investigating HIV risks for dental and medical professionals have been ongoing for years. Then the working environments of HCWs changed dramatically on July 27, 1990. On that date, the CDC reported the first instance of possible HIV transmission from an HIV-infected HCW to a patient.[23] People were stunned. Soon the case involving a Florida dentist was being discussed and debated in many forums. Later reports linking five additional patients to the dentist's HIV strain added to the turmoil and public concern.[24-26]

Unfortunately, the exact mode of viral transmission has not been determined to date. It probably never will.[27,28] Without important epidemiological information, the resultant extensive

media attention and public proclamations of "instant infectious disease experts" have caused some health professionals and the public in general to draw conclusions based more on frustration, fear and anger than on existing scientific data. No matter what happened, the results have already been tragic for those infected, their families and society in general.

Central to the debate surrounding HIV-infected HCWs is what is considered an acceptable level of risk of HIV transmission. The CDC periodically publishes updated retrospective data on patients of HIV-infected HCWs.

In the most recent report, HIV test results have been completed for 19,036 people who were treated by 57 HIV-infected HCWs.[26] The findings from this investigation, and other recently published reports studying HIV-infected physicians and dentists,[29-32] have not found any other HIV transmission from an infected HCW to patients. The Florida dentist remains the only documented case of HIV transmission from a treatment provider to patients.

Some elected officials have attempted to address this issue with proposed legislation that would mandate HIV testing for HCWs, and require those found to be infected to disclose their HIV status to patients. Many who work in the area of infectious diseases see substantial problems with this line of thinking.[33]

Any HCW-to-patient transmission of a bloodborne infection such as HIV or HBV would probably occur only within a narrow set of circumstances. Implementation of broad, potentially prohibitory regulations, therefore, does not appear warranted. At the least, many HCWs who believe they may be at risk for HIV infection would be tested anonymously or avoid testing altogether. As a result, the regulations would succeed only in driving potential problems underground.

The risk of transmitting HIV from patient to HCW is much higher than the risk of transmitting from HCW to patient. For this reason, mandatory testing of HCWs is not defensible without a mandate that patients also be tested routinely. In addition, the period for developing positive serological reactions after HIV infection is long enough to suggest that regular retesting for infection be required.

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UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

DECLARATION OF SANFORD F. KUVIN, M.S., M.D., D.T.M. & H.

Pursuant to 28 U.S.C. § 1746, I, Sanford Kuvin, declare that:

1. I hold a Bachelor of Medicine and Surgery (M.B., B. Chir. Cantab.) with First Class Honors Degree from Cambridge University in England, and I am a licensed M.D. in the United States. I hold a Master's Degree with Distinction in Medical Microbiology from the University of Pennsylvania, and a Diploma in Tropical Medicine and Hygiene with Honors from the London School of Tropical Medicine. I am board qualified in internal medicine, and I am the Founder and Chairman of the International Board of Directors of the Sanford F. Kuvin Centre for the Study of Infectious and Tropical Diseases at Hebrew University Medical School-Hadassah Hospital, Jerusalem, Israel. My education and professional experience are more fully set forth on my Curriculum Vitae, attached hereto.

2. I have reviewed a Curriculum Vitae of David L. Lewis, Ph.D., and I have read David L. Lewis & Max Arens, Resistance of Microorganisms to Disinfection in Dental and Medical Devices, 1 Nature Medicine 956 (Sept. 1995). Based upon my education, experience and expertise I offer the following declaration and

REPORT OF
Sanford F. Kuvin, M.S., M.D., D.T.M. & H.
Re: Sidney Abbott v. Randon Bragdon, D.M.D.

This report is intended to conform with Rule 26(2)(b) of the Federal Rules of Civil Procedure in relation to my views to be presented in the Randon Bragdon, D.M.D. case.

I attach a copy of my curriculum vitae. I also attach a listing of cases in which I have testified as an expert at trial or by deposition within the preceding four years. I will be compensated for my efforts in this matter at a rate of $500 per hour.

The opinions I have in this matter and the reasons therefor follow. I will testify that Dr. Bragdon's "Infectious Disease Policy" is consistent with practicing dentistry in a prudent and safe manner and that the so-called "universal precautions" are not adequately effective in reducing or eliminating the risk of transmitting infectious contagious diseases, including but not restricted to the human immunodeficiency virus, within the dental office setting.

Without limiting the aforesaid, I expect to advance the following opinions.

. . . .

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(9) There exists in the medical literature evidence of clusters of hepatitis B being spread in invasive health care settings without any explanation as to how the transmissions occurred which is a paradigm for HIV cluster spread. The first documented cluster of patients infected with HIV from an AIDS infected dentist similarly remains without any explanation as to how transmissions occurred.

(10) The "universal precautions" are accepted as a standard of practice, but they do not adequately mitigate the small but definite risk associated with the treatment of HIV patients. The

"universal precautions" are unreasonable in respect to Dr. Bragdon because he is unable to deal with all the associated risks of HIV transmission in his office. Dr. Bragdon appears to be a knowledgeable, astute and careful practitioner who is familiar with the assistance offered by the utilization of "universal precautions". However, the barrier techniques available through utilization of "universal precautions" may not be adequate to protect Dr. Bragdon, his patients, his staff, or other patients when utilized in dental treatment in his office.

(11) Treatment of Sidney Abbott, by Dr Bragdon in his office, posed a direct threat as that term is defined under the regulations promulgated to Dr. Bragdon, based on reasonable judgment, relying on current medical knowledge, and on the best available objective evidence, dictates the conclusion that the nature, duration and severity of the risks were such that Dr. Bragdon's office policies were such that no additional modification of his existence of a direct threat. The nature of the risk includes the transfer of the HIV virus or other infectious diseases by blood or bodily fluids in a dental setting. The duration of the risk is life-long with HIV. The severity of the risk is that HIV/AIDS is uniformly lethal. The probability that a potential injury will actually occur is significant.

(12) Treatment with invasive procedures by a physician or dentist or other health care providers infected with HIV poses a significant risk to patients and others such as must disallow such health care provider from proceeding with treatment. The risk is similarly significant for a dentist treating an infected patient in an office setting and should be mitigated by allowing that treatment to be conducted in a setting equipped for the treatment of such patients.

. . . .

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(14) Hepatitis B, another blood-borne virus transmitted identically to HIV, has been documented from patient to health care provider, and from invasive health care provider to patient during

invasive procedures during which "universal precautions" were used.

. . . .

[App. 186]

UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

AFFIDAVIT OF RANDON BRAGDON, D.M.D.

The affiant, Randon Bragdon, D.M.D., being duly sworn deposes and says:

1. My name is Randon Bragdon. I am of legal age and under no legal disability. I have personal knowledge of the facts stated in this affidavit and know them to be true and correct.

2. I hold a Bachelor of Arts in Physics from the University of Maine.

3. After I received my degree in physics, I worked for two years as the training engineer for General Electric. Later, I worked for two years as an electro-optical engineer for Block Engineering, and then started my own electrical contracting business in Maine called Bragdon Electric.

4. In 1975, I began dental school at Tufts Dental School. I graduated from Tufts in 1978 and received my D.M.D. (Doctor of Dental Medicine). In 1978, I received a license in Maine to practice dentistry. Since then, that license has never been suspended nor revoked.

5. I began my private practice of dental medicine in Bangor in 1978. Since then, I have seen over 8,000 patients and have performed thousands of invasive dental procedures, including filling thousands of cavities.

6. When Ms. Abbott visited my office on September 16, 1994, I found that she had a cavity near her gum line on a back, lower, left-side tooth. In technical terms, this would be called a buccal lesion at the cervix of tooth number 19.

7. When I fill a cavity such as Ms. Abbott's, I start by giving one or more injections of local anesthetic into the tissues surrounding the tooth. This process involves grasping the area to be injected with my thumb and stabilizing the patient's head and jaw with my other fingers as I insert the needle beside my thumb, withdraw the needle, and often inject again to ensure complete nerve blockage. During this process, if the patient starts to bite down, which is common, or flinches, I can, and have, stuck myself with the needle just before, between, or after injections, even if I was not careless. Such accidents though relatively infrequent, are inevitable.

8. To remove the decay from a cavity such as Ms. Abbott's, I and other dentists start by using a high speed bur (patients often call it a drill) that turns at 400,000 revolutions per minute. In contrast, a typical high speed electric drill for a home workshop operates at approximately 1200 revolutions per minute.

9. The drilling process usually generates a spattering of blood or bloody saliva, especially when the cavity is next to the gum lime.

10. To keep the tooth from overheating when it is being drilled at such a high speed, my (and other dentists') handpiece emits a continuous cooling spray of water and air. As this spray hits the tooth, gum, and rotating bur, an aerosol mist is generated consisting of tiny droplets of water, blood, and bloody saliva. These droplets remain suspended in the air and drift up into the dentist's face, or they eventually land and remain on other surfaces three to six feet away.

11. Attached to this affidavit is a copy of page 73 of a book called Practical Infection Control in Dentistry (Lea & Febiger 1991) written by James Cottone, Geza Terezhalmy, and John Molinari. John Molinari is one of Ms. Abbott's experts in this case.

12. The narrative indicates that the splashing of saliva illustrated in figures 1-6 is from a single, class-II operative procedure on a mandibular second molar. In layman's terms, this is filling a single cavity on a back, lower tooth, such as Ms. Abbott's. In my experience, having performed hundreds of such procedures, the figures accurately depict the type of splashing of saliva in such procedures. Much of the saliva in such procedures also contains blood in it, and as can be seen, the bloody saliva spreads over many surfaces.

Dated: September 18, 1995 s/s Randon Bragdon, D.M.D.
Randon Bragdon, D.M.D.

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INTERROGATORY NUMBER 4

Describe in detail what occurred when you appeared at Dr. Bragdon's office as is alleged in paragraph 7 of your complaint.

RESPONSE TO INTERROGATORY NUMBER 4

OBJECTION: Ms. Abbott objects to Interrogatory Number 4 because the word "detail" is vague and unclear. Without waiving this objection, Ms. Abbott answers Interrogatory Number 4 as follows:

When I arrived at Dr. Bragdon's office, a receptionist asked me to fill out a medical questionnaire form. The form included questions about whether I was HIV-positive or had AIDS. I filled out the questionnaire and indicated that I was HIV-positive. I returned the form to Dr. Bragdon's receptionist. I then entered Dr. Bragdon's treatment room. Dr. Bragdon examined my mouth by probing with instruments in the general area where I indicated I had a tooth ache--the lower left corner of my mouth. Dr. Bragdon told me that I had a cavity that needed to be filled. Dr. Bragdon also asked me if I had any medical conditions. I told him that I was HIV-positive. He also asked if I had any symptoms. I answered "no." He then indicated that he did not treat anyone with an infectious disease including HIV or AIDS in his office. He said that he would treat me in a hospital. I asked Dr. Bragdon if I would be required to pay for the cost of the hospital. He said that I would be required to pay for the cost of the hospital. I told Dr. Bragdon that I would think about it and get back to him.

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(1) or from the dentist to the six patients did occur in (2) the dental setting: am I correct?
(3) **A** Correct.
(4) **Q** And another conclusion is that you're (5) unable to determine the exact incident or incidents (6) that led to the transmission?
(7) **A** Yes.
(8) **Q** Did the study conclude that the Florida (9) dentist had endeavored to utilize universal (10) precautions?
(11) **A** Yes.
(12) **Q** And did his staff and other witnesses to (13) his involvement and treatment of these patients (14) uniformly support that contention?
(15) **A** The investigation did determine that there (16) were attempts made to follow universal precautions.
(17) The Investigation also determined that these attempts (18) to follow universal precautions were not uniformly (19) adhered to. There were breaches.
(20) **Q** Did the study indicate the breaches were (21) exiguous or that this was a case where there had been (22) no real efforts to follow the precautions or that a (23) careful investigation revealed that there were, on (24) occasion, breaches?
(25) **A** A careful investigation identified

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(1) breaches in infection control. It's safe to (2) characterize that practice as one that did make (3) attempts at compliance with universal precautions.
(4) **Q** There's nothing to indicate, to support (5) the assertion that this dentist failed to follow the (6) universal precautions in the same manner that most (7) dentists attempt to follow it?
(8) MS. PERLEY: Objection as to form.
(9) **A** I don't understand your question. I'm (10) sorry.

(11) **Q** One of the conclusions of the study of the (12) Florida dentist is that the precise method of (13) transmission from the dentist to the patient is (14) unknown, right?
(15) **A** Correct.
(16) **Q** Is it your opinion that in the health care (17) setting, a transmission from a dentist to a patient or (18) a transmission from a patient to a dentist can occur (19) more readily?
(20) MS. PERLEY: Are you talking with respect (21) to an infected --
(22) **A** Can I restate your question to make sure (23) that I'm clear? I believe your question is: Which (24) direction is the transmission of HIV or bloodborne (25) pathogen, which --

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(1) **Q** HIV.
(2) **A** Which direction of transmission of HIV is (3) more likely, a provider who's infected to an (4) uninfected patient or from an infected patient to an (5) uninfected provider?
(6) **Q** That's correct.
(7) **A** The available scientific literature (8) evidence would indicate that the risk of transmission (9) is greater from an infected patient to an uninfected (10) provider.
(11) **Q** How much greater?
(12) **A** That's a complicated answer. I mean, I'm (13) not sure if you want a specific number.
(14) **Q** I want whatever your opinion is
(15) **A** It's greater.
(16) **Q** Why do you think it's greater?
(17) **A** The available evidence would tend to (18) indicate that the risk of using HIV for an example, (19) the risk of HIV transmission from an infected health (20) care provider to a patient is very small. The only (21) case that I am aware of that's reported in the (22) literature today would be the case of the Florida (23) dentist to six patients. There are a number of (24) reports in the literature of health care workers (25) either acquiring or possibly acquiring HIV as a result

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(1) of employment in the health care setting.
(2) We discussed those that are documented (3) versus possible cases earlier. If we would use the (4) numbers that we discussed earlier on possible and (5) documented cases, we have something in the (6) neighborhood of 130 cases total between possible and (7) documented of transmission from patients to providers.
(8) In terms of from provider to patients, currently (9) available information would indicate that that has (10) occurred on six occasions in one practice, so that's a (11) relative comparison of the literature on those small (12) risks.
(13) **Q** When you're referring to a health care (14) provider to a patient, you seem to be referring to (15) dentistry. When you're referring patient to health (16) care worker, you're referring more broadly to the (17) whole health care industry; are you not?
(18) **A** That is because the only report in the (19) literature that I am familiar with of (20) provider-to-patient transmission happens to have (21) occurred in the case of the one Florida dental (22) practice.
(23) **Q** Are you aware of the --
(24) MS. PERLEY: I'm sorry, John. One second, (25) please.

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(1) **Q** Do you have anything you want to add to (2) your answer after your conference with your attorney?
(3) **A** I would like to speak with counsel.
(4) **Q** Sure.
(5) Whereupon discussions ensued between the (6) witness and Ms. Perley outside the hearing of the (7) court reporter.)
(8) **A** No.
(9) **Q** When you're referring to the statistics (10) that you've just described, I gather you're referring (11) to the United States and not foreign countries; is (12) that correct?
(13) **A** Actually, I'm referring to the available (14) information, and that is foreign countries as well.
(15) **Q** Actually, I was trying to define (16) specifically the 42 cases, if that's the number. Did (17) they all occur in the United States?
(18) **A** No. It's my understanding that the (19) occupational transmission cases represent cases (20) principally reported in the

United States, but I (21) believe there are some cases that are from a couple of (22) other countries reported in there as well.
(23) **Q** So, it's mostly from the United States?
(24) **A** It's principally from the United States, (25) yes.

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PATIENT REGISTRATION AND HEALTH RECORD

In order to help me render the proper dental services to you, would you please be kind enough to answer the following questions. Please note the space for remarks for any answers that require clarification or any other information you think I should have. Thank you for your cooperation.

Date: 9-16-94
Name: Sidney Abbott Date of birth: 1-3-61 Marital Status Divorced
Address: Phone: 989-7017
Street: 16 State St Apt E
City: Brewer State: ME Zip: 04412
Referred By: A friend
Employed By: Penobscot Area Housing Position: Dir. Serv. Worker
Business Address: 215 French St Bangor Phone: 942-3994
Spouse_____Date of birth_____
Spouse Employed By_____Position_____
Business Address_____Phone_____
In case of an emergency,
who should be notified: Melinda Perrier Phone: 947-2367
Person responsible for this account: me
Social Security #: 006-68-6050 Spouse's SS#_____
If you have insurance, name of insured_____
If your spouse has insurance, name of insured_____
Insurance Company_____Policy #_____

Medical Health

General Health (please check): Good
Are you presently under the care of a physician: Yes
What medications are you presently taking?_____
Physician: Rob Pinskey Last complete physical: Jan
What disorders are you presently being treated or have been treated for within the past year? HIV
Please list any serious illness within the past five years, not listed above._____
Have you been told by a physician that you should have prophylactic antibiotics before having dental procedures done? No

General Health

	Yes	No		Yes	No
Tire easily		x	Persistent Cough		x
Headaches	x		Sputum (phlegm) production		x
Night sweats		x	Cough up bloody Sputum		x
Persistent fever		x	Marked weight change		x
Glaucoma		x	Shortness of breath		x

Have you been diagnosed or treated for:

	Yes	No		Yes	No
Heart Disease		x	Ulcers		x
Asthma or hay fever		x	Heart murmur	x	
Rheumatic or Scarlet fever		x	Jaundice		x
Abnormal blood pressure		x	Hepatitis		x
Congenital heart lesions		x	Sinus trouble		x
Tuberculosis or lung disease		x	Cough		x
Diabetes		x	Arthritis		x
Epilepsy		x	Stroke		x
Anemia		x	Glaucoma		x

[App 236]

tions by his [17] staff. And yet, this cluster of cases of HIV [18] occurred in Florida. [19] We also know conversely that health [20] care workers have been infected, because blood is a [21] two-way street. And it's not surprising, [22] therefore, that health care workers have been [23] infected from patients. And the CDC lists health [24] care workers that have been so infected with HIV.

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[1] The official numbers, as you know, are about 42; [2] but there are an additional 91 which are also on [3] the list, six of which are dentists.
[4] Having said that, we know that gloves [5] rip, tear, leak. They're porous. They have many [6] defects to them. And there is not much evidence to [7] show in the literature that a reduction in disease [8] has occurred of great significance since the [9] introduction of universal precautions with barrier [10] techniques. [11] However, I said before, I'm a great [12] advocate of it, because medical logic suggests that [13] using barriers will prevent disease transmission. [14] In trying to prevent the transmission of HIV in the [15] dental operatory, the use of gloves, the washing of [16] hands in between patients, the double gloving where [17] possible, are all methods of universal precautions [18] to reduce the risk, although the risk is still [19] there. It's significant. It's real. It's low [20] risk. But it's certainly absolutely real. [21] And in trying to reduce the risk, the [22] dentist should wear these gloves. And you asked [23] how can this protect. But unfortunately the [24] dentist, as we know, will stick himself or herself

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[1] no matter how hard he or she will try to not stick [2] him or herself. And as you well know, the [3] literature is replete with studies to show that no [4] matter what care responsible invasive health care [5] workers take, they will and are guaranteed to stick [6] themselves, because, after all, they're making [7] human errors.

[8] With regard to other methods of [9] prevention, utilizing universal precautions; the [10] sterilization of instruments; the sterilization of [11] hand pieces, even though there's no evidence that [12] the hand piece has ever transmitted HIV, have been [13] put into use. And in fact many recommendations of standard of care, [15] many of their recommendations have been made based [16] on medical logic and not necessarily on hard [17] science. [18] There's nothing in hard science to [19] show that sterilizing hand pieces prevents the [20] transmission, or indeed there's been no [21] transmission that's been documented of HIV through [22] an unsterile hand piece. But yet the Centers for [23] Disease Control says the standard of care should be [24] to sterilize all hand pieces. No science behind it

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[1] but medical logic.
[2] And this whole discussion, and please [3] accept this, is based on medical logic. Nothing [4] more, nothing less. And the problem with dealing [5] with something of this terribly important nature, [6] namely the health of the uninfected, the rights of [7] the uninfected, the right to know, the duty to [8] warn, are all based on medical logic.
[9] Now, to continue with my answer on [10] your question about protecting, using universal [11] precautions, the use of masks is certainly in [12] order. Does it prevent the spread of HIV from the [13] patient to the health care worker? There is no [14] evidence that suggests that. However, the CDC in [15] its wisdom says wear a mask. Again, medical [16] logic. Why? Because there's no evidence to [17] suggest that aerosol spray can transmit HIV. But [18] do they know for certain? No.
[19] In fact, if one looks at the very [20] recent issue of the Morbidity and Mortality Weekly [21] Report of just a week and a half ago, you'll find [22] an update on viral hemorrhagic fever. Ebola virus. [23] And it's updated for the United States. And what [24] it says is that there is no evidence of aerosol

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[1] transmission of Ebola fever; however, don't assume [2] that to be totally correct, because we know that [3] moneys can do it by aerosol.
[4] We know that there's no evidence to [5] suggest that it can occur in man, but be aware and [6] take precautions against it. So it merely is a [7] paradigm of what we don't know and what we're [8] searching for. And I hope and I pray that all of [9] us are searching for the better health of the [10] American public.
[11] **Q:** When you refer to science, you're [12] referring to cases that have been documented; is [13] that right?
[14] **A:** That's correct.
[15] **Q:** Do you believe the CDC is a reliable [16] authority on the transmission of HIV?
[17] **A:** Certainly I believe the CDC is a reliable [18] authority on the transmission of HIV.
[19] **Q:** You mentioned that giving birth is a mode [20] of transmission of HIV; is that right?
[21] **A:** That's correct. Please let me say that [22] it would have to be from an HIV-infected mother to [23] a newborn.
[24] **Q:** So if you're female HIV positive there's

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[1] a substantial risk that your child will be HIV [2] positive?
[3] **A:** There is indeed.
[4] **Q:** And you mentioned the exchange of blood [5] and bodily fluids is another mode of HIV [6] transmission?
[7] **A:** That is correct.
[8] **Q:** Which bodily fluids are you referring to?
[9] **A:** Vaginal secretions; semen; bodily fluids [10] of the abdominal cavity where a surgeon has his or [11] her hands in the cavity and there's an exchange of [12] fluids; the oral cavity, of course, if there are [13] cuts, abrasions; the mucocutaneous entrance. [14] That's not an exchange, but the mucocutaneous [15] surfaces such as the eye, rectum, can receive the [16] virus.
[17] Saliva contains the virus, of course, [18] but by most authorities not in quantitative amounts [19] that are readily transmitted, unless mixed with [20] blood. And as you may know, there have been cases [21] of transmission through sexual acts of cunnilingus [22] which have been documented by the CDC. So

whether [23] that's pure saliva or saliva mixed with other [24] things has yet to be determined. But there are,

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[1] for the record, examples of cunnilingus where HIV [2] has been transmitted.
[3] **Q:** Is your opinion in this case based on the [4] assumption that saliva is a mode of transmission [5] from an HIV-positive person to another person of [6] HIV?
[7] **A:** I said there's been no evidence to [8] suggest on the face of it that saliva unattended, [9] namely, not mixed with blood, has been a method of [10] transmission. I would have to add parenthetically, [11] however, that the virus does exist in saliva, but I [12] would also say not in quantitative amounts that [13] have been shown to be transmissible. And, just for [14] the record, there are some anti-HIV constituents of [15] saliva which may negate against the virus itself.
[16] **Q:** And it's your understanding that sweat is [17] not a bodily fluid that transmits HIV?
[18] **A:** That's correct. However, sweat is a [19] bodily fluid that can transmit hepatitis B.
[20] **Q:** So in the dental office the primary body [21] fluids by which HIV could be transmitted would be [22] blood is that right?
[23] **A:** Yes. And as I said, and I've already

EXCERPTED FROM THE AMERICAN DENTAL ASSOCIATION POLICY ON AIDS, HIV INFECTION AND THE PRACTICE OF DENTISTRY

PROFESSIONAL CONSIDERATIONS

The Association believes that HIV-infected individuals should be treated with compassion and dignity. Current scientific and epidemiologic evidence indicates that there is little risk of transmission of infectious diseases through dental treatment if recommended infection control procedures are routinely followed. Patients with HIV infection may be safely treated in private dental offices when appropriate infection control procedures are employed. Such infection control procedures provide protection both for patients and dental personnel.

A sound approach to the treatment of infectious patients requires an assessment of the patient's condition based on reasonable and informed medical judgments, given the state of medical knowledge at the time. A dentist should not refuse to treat a patient whose condition is within the dentist's current realm of competence solely because the patient is HIV infected.

Dentists should be alert to signs and symptoms of HIV infection that may be identified during the provision of dental care. Patients with histories or conditions possibly indicative of HIV infection should be referred to their physicians for diagnostic procedures, counseling and medical follow-up.

The Association believes that all HIV-infected patients should disclose their HIV status and medical history; dentists, like physicians, need to know every patient's medical history in order to make appropriate treatment decisions that are in the best interests of the patient. For the same reason, the Association also strongly supports state and federal legislation that gives an HIV-infected patient's health care providers the right to share knowledge of the patient's HIV status and current medical condition without risking a violation of state or federal antidiscrimination laws and HIV confidentiality laws. The Association further strongly supports state

and federal legislation that protects a dentist from charges of discrimination if a dentist, in a sincere effort to protect a patient's health, elects to refrain from treating a patient who failed to disclose his/her HIV status and medical history or allow an HIV test to be performed.

The Association urges dentists to maintain strict confidentiality of the patient's HIV status and medical condition, except as noted above with respect to sharing of information among the patient's health care providers when allowed by state or federal law. Dentists are encouraged to have an office protocol for the confidential handling of information about an HIV-infected patient.

The Association believes that individuals with HIV infection should have access to dental treatment. Treatment considerations should provide for a judicious balance between the well-being of these patients and the protection of the health of the public as well as the dental care providers.

(TRANS. 1991:591).

The publication of this document was supported by the ADA Health Foundation, with an educational graph provided by the Werner-Lambert Company.

[App. 273]

UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

AFFIDAVIT OF ARLEEN RICHMAN

The affiant, Arleen Richman, being duly sworn deposes and says:

1. My name is Arleen Richman. I am of legal age and under no legal disability.

2. I am the Director of Communications of the United States Parachute Association (USPA), 1440 Duke Street, Alexandria, VA 22314. In my capacity as such, I have personal knowledge of the facts stated in this affidavit.

3. Since 1984, it has been the regular practice of the USPA to compile the statistics concerning the number of deaths per jump that are contained in the attached table for years 1984 to 1992. These statistics are derived from information received from people with knowledge of the relevant numbers of jumps and associated skydiving fatalities, and who keep such information in the regular course of their skydiving associations. The statistics concerning the number of deaths per jump for years 1984 to 1992 were compiled at or near the time such information was received.

Dated: September 29, 1995 S/Arleen Richman
Arleen Richman

STATE OF VIRGINIA
COUNTY OF ALEXANDRIA, ss. Date: Sept. 29, 1995

Personally appeared the above-named Arleen Richman, who first being duly sworn, made oath that the foregoing affidavit which she has signed and to which she subscribed is based on her personal knowledge and that the facts set forth therein are true.

Before me,

/s/ BJ

Name: BJ

Notary Public

My Commission Expires September 30, 1999

[App. 351]

SKYDIVING AT A GLANCE

United States Parachute Association (USPA)
(703) 836-3495

WHO SKYDIVES? You're in good company -- USPA membership is slightly over the 29,000 mark and growing by about 5% a year. About 12-15% of our members are women. More than 90% of our members have a high school diploma; 40% are college graduates. Members range in age from 16 -- (permissible at some Drop Zones) to well into their 70s and 80s.

CAN ANYONE JUMP? Almost anyone can successfully complete a first jump. No experience is required. Individual skydiving centers, called Drop Zones or DZs, set their own guidelines. Generally, you need to be in good health, age 18 (16 with parental consent, if DZ policy allows) and weigh no more than 225 pounds.

HOW DO I START? USPA recognizes 3 types of first jumps:

Tandem: Student is attached to the front of the Instructor's harness and is carried as a passenger throughout the jump from exit until landing. Requires minimal ground school.

Static Line: You exit solo from the aircraft. Your chute is automatically deployed as you exit the plane at about 3,000 feet. Requires ground school and attaining learning objective.

AFF: (Accelerated Freefall) Two Jumpmasters exit the aircraft holding on to you (one on each side) and assist you while you freefall for about 45 seconds. Once you open the parachute, the Jumpmasters leave and you make a solo descent. Requires classroom instruction prior to first jump. This training program progresses through 7 levels of instruction, at which time you should be well on the way to meeting the requirements for your "A" license.

WHERE: At a Drop Zone that is a Group Member of the USPA. USPA Group members have pledged to follow USPA Basic Safety Requirements and Recommendations for student and advanced skydivers and to offer first-jump courses taught by USPA rated instructors. See the attached listing for the Centers nearest you.

HOW MUCH: Expect to pay between $150 and $200 for a first tandem or static line jump. First time freefall costs between $250 and $300. Fees include all equipment and training. Experienced skydivers (those who jump without instruction) can jump for between $6 and $20 depending on type of aircraft used and altitude of exit.

THE RISK: Parachuting has an inherent risk but new and modern equipment, improved techniques, and USPA certified instructors have sharply reduced most of the dangers. Annual rate of fatalities is usually between 26 and 30 or approximately 1.2 in 100,000 jumps.

TO ENHANCE YOUR SAFETY:
*Plan to make your first jump at a USPA Group Member *Ask to see the instructor's USPA rating *Use an Automatic Activation Device (AAD) required by USPA's Basic Safety Requirements *Check out the Drop Zone BEFORE you jump. How experienced are the pilots and how well maintained are the planes? What feeling/attitude do you get when you visit the DZ? Does safety seem to be everyone's first concern?

Blue Skies!!!!!! Hope to see you up there soon!!
Any Questions--call 703-836-3495

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RIS

STATISTICS 10/93

Although skydiving is statistically a safe sport, it is inherently dangerous. With the proper training and equipment the risk can be minimized.

Year	Participants	Jumps	Fatalities	Deaths per jump*	Deaths per participant*
1983	101,500	UKN	29		28.5
1984	100,300	2,373,000	33	1.2	32.9
1985	102,100	2,329,000	26	1.1	25.5
1986	105,600	2,246,000	30	1.3	29.4
1987	108,200	2,289,000	28	1.2	25.9
1988	110,700	2,297,000	23	1.0	20.8
1989	111,880	2,352,000	36	1.5	32.2
1990	115,500	2,400,000	23	.9	19.9
1991	121,900	2,440,000	30	1.2	24.6
1992	136,528	2,600,000	27	1.0	19.7

5 YEAR AVERAGE

YEARS	PARTICIPANTS	FATALITIES	DEATHS PER PARTICIPANTS*
87-91	113,636	28	24.6
82-86	104,250	29.4	28.2
77-81	108.940	49	44.9
72-76	99,000 est	40.6	41.0 est

1991 death rates:

All accidents 34.9*
motor vehicles (ages 25-44) 32.0*
in 33 states the accidental death rate is greater than 35 deaths per 100,000 population

*based on ratio of deaths per 100,000 (jumps or participants)

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

Food and Drug Administration
Bethesda MD 20892

Date: April 23, 1992

From: Director, Center for Biologics Evaluation and Research

Subject: Revised Recommendations for the Prevention of Human Immunodeficiency Virus (HIV) Transmission by Blood and Blood Products

To: All Registered Blood Establishments

INTRODUCTION

On September 25, 1991 the Food and Drug Administration (FDA) licensed the first enzyme immunoassay (EIA) for the simultaneous detection of antibodies to human immunodeficiency virus type 1 (HIV-1) and type 2 (HIV-2) in human serum and plasma. This test, manufactured by Genetic Systems Corporation, is based on whole viral lysate antigens. Another combination EIA, based on recombinant antigens, and manufactured by Abbott Laboratories, received a license on February 14, 1992. These tests are indicated for detection of antibodies to HIV-1 and/or HIV-2 in human serum or plasma. The tests may be used for donor screening and as an aid in the diagnosis of potential infection with HIV-1 and/or HIV-2.

The availability of licensed screening tests for combined detection of antibodies to HIV-1 and HIV-2 provides blood centers with the capability of performing routine HIV-2 screening without the need to implement a second test in addition to the currently required test for antibodies to HIV-1. Based on this possibility, the issue of whether to recommend donor screening for HIV-2 was discussed at a meeting of the Blood Products Advisory Committee on September 27, 1991.

At the time of the meeting, only 31 cases of HIV-2 infection in the U.S.A. had been reported to the Centers for Disease Control, despite extensive surveillance studies. (One additional case has been reported since then.) These data indicated that HIV-2 infections presently represent only a minimal risk to the safety of the blood supply. Nevertheless, from a public health perspective, use of combination tests offers an opportunity to further protect the safety of blood recipients against HIV-2 infection without requiring an increased number of tests in blood centers. Donor screening for antibodies to HIV-2 would also reduce the need for continued surveillance studies which are expensive, difficult to sustain, and might nevertheless fail to prevent the earliest cases of HIV-2 transmission by blood products.

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For these reasons, and with the concurrence of the Blood Products Advisory Committee, the FDA recommends that all establishments collecting whole blood, blood components, Source Plasma or Source Leukocytes implement a licensed test for detection of antibodies to HIV-2 by June 1, 1992. For this purpose the Agency finds acceptable either the use of a licensed combination test for detection of antibodies to HIV-1 and HIV-2 or the use of two separate tests licensed for detection of these antibodies.

To implement HIV-2 testing, modifications are necessary to the current recommendations for the prevention of HIV transmission by blood and blood products. The modifications affect donor selection and deferral procedures, the HIV reentry algorithm, Public health Service recommendations for additional medical follow-up and counseling, and the management of potentially infectious units from prior collections. For this reason, the FDA is issuing a revised set of recommendations which replaces the Agency's memoranda to blood establishments dated February 5, 1990 and December 5, 1990.

The FDA further discussed donor deferral criteria for HIV at a meeting of the Blood Products Advisory Committee on March 13, 1992. Based on updated scientific data which were presented at this meeting, modifications have been made to some of the deferral

criteria. The changes include a 12 month instead of lifetime deferral for sexual partners of persons with high risk behavior, and voluntary instead of recommended use of Confidential Unit Exclusion. Also, the FDA is discontinuing its recommendation for documentation in the donor record of the medical history of HIV associated signs and symptoms.

2

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[App. 355]

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B. Criteria for the Exclusion of Unsuitable Donors Who Are at Increased Risk for HIV

The minimum information presented to potential blood and plasma donors at every visit should indicate clearly that persons meeting any of the following descriptions or having engaged in any of the following activities should not donate blood or blood components to be used for transfusion or further manufacturing:

- Persons with clinical or laboratory evidence of HIV (AIDS virus) infection.[1]
- Men who have had sex with another man even one time since 1977.
- Past or present intravenous drug users.
- Persons with hemophilia or related clotting disorders who have received clotting factor concentrates.
- Men and women who have engaged in sex for money or drugs since 1977.

[1]At this date, the most recent criteria from the CDC appear in MMWR 1987;36 (S-1):1-9. A revision of these criteria is anticipated in the near future.

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* Persons who have had sex with any person meeting the above descriptions during the preceding 12 months.

* Persons who have had, or have been treated for, syphilis or gonorrhea during the preceding 12 months, or who have had a reactive screening test for syphilis in the absence of a negative confirmatory test in the last 12 months. (Any synonyms for these diseases that may be appropriate to the local donor population should be included in the donor questionnaire and oral interaction.)

  [Note: Refer to FDA's memorandum to all blood establishments dated December 12, 1991 for additional discussion.]

* Persons who have received a transfusion of whole blood, a blood component (e.g. cryoprecipitated AHF, platelets) or a clotting factor concentrate (e.g., Factor IX, Human) within the past 12 months. Receipt of an FDA-licensed plasma derivative other than a clotting factor concentrate (e.g., Albumin [Human]) is not a basis for exclusion.

* Persons born in or emigrating from countries where heterosexual activity is thought to play a major role in transmission of HIV-2 infection (i.e. sub-Saharan Africa and islands located near these areas of Africa) and persons who have had sex with any person meeting the latter description.

  [Note: These criteria should be discontinued upon implementation of a test for antibodies to HIV-2 no later than June 1, 1992.]

Sample direct questions on behavior related to increased risk of HIV infection are provided as an Addendum to this document.

During the health history interview, a donor may volunteer information about a specific instance of possible exposure to hepatitis viruses or to HIV. Suggested deferral periods for two such instances are provided below:

* Persons who have been victims of rape during the preceding 12 months should be deferred.

* Persons who have had contact with blood and body fluids through percutaneous inoculation (such as

injury or accidental needlestick) or through contact with an open wound, non-intact skin, or mucous membrane during the preceding 12 months should be deferred.

Donors should also be informed that:

* There is a time interval early in infection during which tests for HIV may be negative although an infection may still be transmitted.

* A sample of blood will be tested for antibodies to HIV and the donor will be notified if a test is positive (see I.C.1.c.); individuals with positive tests will be permanently deferred from future blood or plasma donation.

* The names of people with repeatedly reactive tests for anti-HIV-1 or anti-HIV-2 will become part of donor deferral registries [as required by 21 CFR 606.160(e)].

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UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

SECOND DECLARATION OF
SANFORD F. KUVIN, M.S., M.D., D.T.M. & H.

Pursuant to 28 U.S.C. § 1746, I, Sanford Kuvin, declare that:

1. I hereby incorporate as if fully set forth herein ¶ 1 of my September 14, 1995, Declaration in this action. Based upon my education, experience and expertise, I offer the following declaration and opinions.

2. Both the blood and the saliva of an HIV-positive patient contain the human immunodeficiency virus (HIV). There is no scientific evidence that a dentist can become infected with HIV through contact only with saliva from an HIV-positive patient. However, there is scientific evidence that a dentist can become infected with HIV through contact with blood or bloody saliva from a patient infected with the virus that causes AIDS. Transmission of HIV from a patient to a dentist can also certainly occur, for example, if a drop of bloody saliva, in splatter or in aerosol form, makes contact with a mucous membrane of the dentist such as the eye, the nose or the throat.

3. Page five of Ms. Abbott's brief quotes page 55 of my deposition out of context and thereby tries to suggest that no aerosol transmission of HIV is possible. That is patently false,

since it does not differentiate between blood or bloody saliva in aerosol form, and saliva unmixed with blood, in aerosol form. The

context of my deposition, pages 55-59, and especially pages 58-59, makes this clear.

4. Page eight of Ms. Abbott's brief wrongly implies that there is no risk of HIV transmission from patient to patient in the dental office. In view of current technologies, the transmission of viruses and bacteria can occur through the transmission of bloody saliva that has retracted into a water line and then passed to a subsequent patient. In addition, transmission can occur through infrequent, but inevitable break downs in universal precautions, as, for example, when a previously contaminated needle, handpiece, or other instrument is not properly sterilized or disposed of. Universal precautions, when properly and thoroughly employed, help to reduce the risk of transmission of HIV, but they do not eliminate the risk.

5. Universal precautions simply do not equate with universal safety. The inexplicable transfer of clusters of hepatitis B virus ("HBV") to patients that have occurred during invasive medical procedures when universal precautions were diligently employed attest to this fact. HBV is a bloodborne virus spread by identical methods as HIV.

6. The recently published article by David Lewis and Max Arens in the prestigious peer reviewed journal Nature Medicine (attached as Exhibit 7 of Dr. Bragdon's Motion for Summary Judgment) indicates that the risk of transmission of HIV from patient to patient in dental offices may be greater than many experts had previously suspected.

7. Tuberculosis (TB) is spread casually, by droplet infection.

8. The CDC states in the attached excerpt from MMWR that HIV infection is the strongest risk factor yet identified for the development of TB disease in persons having TB infection.

and that all HIV-infected persons should receive a PPD-tuberculin skin test in settings where HIV-infected persons receive care.

9. The CDC recommends that all persons with HIV be suspected of having TB and that they may be referred to an appropriately engineered facility (e.g., one with ultraviolet germicidal irradiation and high-efficiency particulate air filtration (HEPA)) for dental treatment involving cough-inducing or aerosol-generating procedures.

10. The CDC's recommendations regarding TB and HIV in the dental setting are, if not logically inconsistent, certainly are in tension with one another. This tension may be the result of (i) the political and civil rights atmosphere surrounding HIV that has led the CDC to downplay the risk of transmission of HIV, and (ii) the absolutely non-political, non-civil rights atmosphere of tuberculosis. However, since the CDC's recommendations regarding TB in the dental setting are of more recent origin than the CDC's last official pronouncement regarding HIV in the dental setting, it may also be the case that the CDC is finally in the process of beginning to deal more responsibly with the gravity of the issue of HIV in the dental setting. TB is a reportable disease in Maine, but HIV is not.

11. Based upon and consistent with CDC recommendations regarding TB, Dr. Bragdon could, as a general rule, decline to perform cough-inducing or aerosol-generating dental procedures (such as filling a cavity) on any HIV positive person in his office on grounds that he or she can reasonably be suspected of having active TB and should receive such treatment in an appropriately engineered facility, as recommended by the CDC.

12. Based upon the conflicting recommendations of the CDC regarding HIV in the dental setting, a dentist might also reasonably decide to discount the possibility of active TB in an HIV-positive person who does not seem infectious with active TB.

A more conservative approach, however, would be for the dentist to adopt a general policy of referring all cough-inducing, or aerosol-generating, procedures on HIV-positive patients to an appropriately engineered TB facility. This is because, among other things, it is quite difficult to diagnose active TB in a person with HIV. People with HIV often show false negatives for active TB when given the customary tuberculin purified protein derivative (PPD) test. Even chest x-rays often fail to reveal active TB in a person with HIV. And simply because a person looks healthy and is not coughing does not mean that she does not have active TB.

13. Because it is difficult, if not impossible, for a dentist in his office to diagnose active TB in a person with HIV, and because people with HIV are at high risk for active TB, the CDC's recommendation that all people with HIV who are to undergo cough-inducing aerosol-generating dental procedures should be considered infectious with TB, makes medical sense, as

does the automatic referral of such cases to an appropriately engineered dental facility.

14. To the best of my knowledge, no CDC publication has ever concluded that dentists should perform invasive dental procedures on HIV-positive patients in the office setting. Instead, the CDC has carefully limited itself to the unremarkable conclusion that such procedures "can" or "may" be performed safely with the use of universal precautions, and that additional precautions are therefore "unnecessary."

15. To the best of my knowledge, no CDC publication, or any reliable, published authority, has ever concluded that the risk of transmission of HIV from patient to dentist using universal precautions during an invasive procedure is infinitesimal. The CDC characterizes the risk as "small."

16. Because of the dearth of comprehensive, scientific studies on the issue, some authorities conclude that the risk of transmission of HIV from patient to dentist cannot be accurately quantified. While this is arguably true, some fairly reasonable estimates can nevertheless be constructed from available, although limited, data. For example, the average dentist performs approximately 3,000 invasive procedures per year (in contrast to 500 for the average surgeon), and Dr. Greenspan reports an average of 1.5 needle-stick injuries per 100 injections given by dentists. See Greenspan Dep. At 156-159. The CDC now estimates that the rate of fatal outcomes from percutaneous injuries causing contact with contaminated blood is

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approximately 3 per 1000. Not all needle-stick injuries to dentists result in parenteral exposure to a patient's blood. Assuming only 1 in 3 such injuries result in such exposure, the risk of a dentist receiving a fatal needle-stick while giving one injection to a single HIV-positive patient is (1.5/100) x (1/3) x (3/100), which is equal to 4.5/300,000, which is equal to 1/66,667. According to calculations provided to me by Professor Halteman (attached), this means that if a dentist practices for 30 years, and gives an average of 235 injections per year to HIV-positive patients, that dentist has more than a 10% chance of sustaining at least one fatal, HIV needle-stick injury during that time. In addition, if the dentist gives only 5 injections per year to HIV-positive patients for 30 years, the dentist has more than a 50% chance of sustaining at least one percutaneous exposure to HIV-infected blood, which is a life-threatening event.

17. The method employed in the previous paragraph to estimate the risk to dentists from accidental needlesticks accords entirely with the method employed by Dr. David Bell from the CDC to estimate the risks to patients from HIV-infected surgeons. The Fourth Circuit relied upon Dr. Bell's estimate in a case called Doe v. University of Maryland Medical Systems Corp., 50 F.3d 1261, 1263 (4th Cir. 1995). I served as an expert witness on behalf

of the hospital in that case. We know that the dental profession is more invasive than the practice of most surgeons, and that blood is a two-way street.

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18. The estimate in the next to the preceding paragraph does not take into account the additional risk of percutaneous injuries to dentists caused by the dental bur and other sharps. Nor does it take into account the additional risk of exposure to blood or bloody saliva in aerosol form that penetrates mucous membranes or nonintact skin. The estimate in the preceding paragraph is therefore a conservative estimate.

19. Dr. Craven is wrong in his Affidavit, ¶¶ 7-10, to imply that somehow the risk of transmission of HIV from patient to dentist is less than one in a billion. This analysis is severely flawed for several reasons.

a. Of his estimated 1.4 billion to 2.8 billion dental procedures in the United States since 1982, only a small percentage of them were performed on people with HIV. It is patently fallacious to imply that the risk of transmission of HIV from patient to dentist is low because patients without HIV have not transmitted HIV to their dentists.

b. Dr. Craven incorrectly states that the CDC has monitored the occupational transmission of HIV from patients to dentists. When a person with HIV goes to a dentist, knowingly or unknowingly, it is not reported to the CDC. When a dentist sticks himself with a needle, or has a sharps injury, it is seldom reported to the CDC. Various institutions do have rules of the reporting of needlesticks, and studies have shown that underreporting of needlestick injuries range from 70% to 95%. See AIDS Clinical Care, Vol. 5, No. 10, p. 78 (Oct. 1993). The CDC has also cited 1983 and 1991 studies for the view that as few

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as 10% of percutaneous injuries are reported. See Ex. 1 of Dr. Bragdon's Motion for Summary Judgment, p. 320.

One of those studies cited by the CDC, which is a reliable authority found in the American Journal of Medicine, is attached. The authors state that failing "to report exposures to HIV-infected blood leads to an inaccurate estimation of prevalence and a subsequent underestimation of the overall occupational risk of acquiring HIV infection" and that the "reported circumstances of exposure suggest that compliance with current infection-control procedures will not prevent many needlestick injuries." Based on their data, the authors extrapolated that approximately 1 in 1,000 medical interns will become infected with the AIDS virus during only 12 months of working in hospitals as a result of accidental needlesticks.

OSHA regulations require dentists to keep records of sharps injuries suffered by their employees, but the regulations do not require that the records be sent to OSHA, much less to the CDC. See 29 CFR § CFR 1910.1030(h)(3).

c. In reality, what the CDC euphemistically refers to as its "passive surveillance system," Ex. 1 of Def.'s Mot. For Summ. J., p. 320, is that every once in a while a dental worker who has reason to suspect he or she has been infected by an accidental exposure to HIV-infected blood has, for whatever reason, decided to contact the CDC. According to page 1238 of a CDC article in the September 1995 issue of JADA (attached as Ex. 1a to Dr. Bragdon's opposition memorandum), this type of voluntary disclosure has happened 19 times. Then, if the dental worker is

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immediately tested for HIV and is negative, and later tests positive in one to six months, and has no other risk factors, he or she

becomes a documented case of HIV transmission. If he or she has no other risk factors, but is not immediately tested and then tests positive in one to six months, he or she becomes a "possible" case of occupational transmission. There have been 6 such "possible" occupational transmissions to dental workers out of only 19 reported exposures. See id., Ex. 1a at 1240. Thus, almost 1/3 of all reported exposures for dental workers are cases in which dental workers have HIV, and no other identifiable risk factors apart from accidental occupational exposures! With other non-politically charged infectious diseases such as hepatitis B, the practice in the medical profession has been to treat analogous "possible" cases as actual cases, and to make policy accordingly.

d. Dr. Craven's analysis, which is based only upon the lack of any CDC documented occupational transmission of HIV to a dentist, necessarily overlooks the risk of HIV transmission from blood, or bloody saliva, in aerosol form. In fact, there are over twice as many CDC reported cases of HIV-infected health care workers without any identifiable non-occupational risk factors, but whose source of HIV-infection is officially unknown (91 such cases), than there are CDC documented cases of occupational transmission of HIV to health care workers (42 such cases).

20. Ms. Abbott's brief on page 11 wrongly implies that I believe Ms. Abbott would have a ten percent risk of acquiring an infection just from having her cavity filled as an outpatient in

a hospital. The ten percent figure discussed during my deposition is for patients who are in a hospital for a prolonged stay, and not for the relatively brief stay of a dental outpatient visit.

21. As a matter of medical ethics, I believe that providing routine dental care to HIV-positive patients is not so inherently dangerous that dentists should not be required to provide such care. However, page 11 of Ms. Abbott's brief incorrectly implies that I believe Dr. Bragdon should be compelled to treat

Ms. Abbott in his dental office. The context of my statement during my deposition, pages 188-189, shows that I also believe as a matter of medical ethics, that Dr. Bragdon should have the right to exercise dental and medical judgment about where and how to treat his patients in a professionally confident, comfortable, and comprehensive setting. As a matter of law, because of the significant risk to Dr. Bragdon, I believe that he cannot and should not be compelled against his will and judgment to perform such invasive procedures in his office.

I hereby declare under penalty of perjury that the foregoing information is true to the best of my knowledge and belief.

Executed on October 4, 1995.

S/Sanford F. Kuvin
Sanford F. Kuvin, M.D.

Sanford F. Kuvin, M.D. did personally appear before me on this day and being personally known to me.

Official Notary Seal
Mollie Gutierrez
Commission Number
CC2024980
My Commission Exp.
June 21, 1996

S/Mollie Gutierrez
Mollie Gutierrez, Notary Public

UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

DECLARATION OF WILLIAM A. HALTEMAN, Ph.D.

1. I hold a Ph.D. in Biostatistics from the University of Washington, Seattle. I have taught Statistics at the University of Maine for 15 years.

2. Based upon my education, experience and expertise, I offer the following declaration.

3. Assuming (a) the odds are 1.5/100 that a dentist will sustain an accidental needlestick while giving a single injection to a person with HIV; (b) if a dentist sustains an accidental needlestick, the odds are 1/3 that he will have parenteral exposure to the patient's blood; (c) if a dentist has parenteral exposure to a patient's HIV-infected blood, the odds are 3/1000 that he will become infected with HIV; and (d) each injection, accidental needlestick, and exposure is a logically independent event, then the odds are (1.5/100) x (1/3) x (3/1000), which is equal to 1/66,667, that the dentist will become infected with HIV as a result of giving a single injection to a person with HIV.

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4. Assuming (a) through (d) in the preceding paragraph, if the dentist gives 7024 injections to persons with HIV, then he will have a 10.000025% chance of becoming infected with HIV on at least one occasion. 7024 divided by 30 is 234.13, which means that if the dentist practices for 30 years, and gives an average of 235 injections per year to HIV-positive patients, the dentist will

have a slightly greater than 10% chance of becoming infected with HIV on at least one occasion.

5. Assuming (a) through (d) in Paragraph 3, a dentist has a (1.5/100) x (1/3), or 1.5/300, chance of having a parenteral exposure to the patient's blood as a result of giving a single injection to the patient. If the dentist gives 139 injections to persons with HIV, then he will have a 50.18% chance of having at least one parenteral exposure to HIV-infected blood as a result of giving the injections. 139 divided by 30 is 4.63, which means that if the dentist practices for 30 years, and gives an average of 5 injections per year to HIV-positive patients, the dentist will have a slightly greater than 50% chance of having at least one parenteral exposure to HIV-infected blood.

6. Attached hereto is a brief narrative discussion of some of the general principles involved in these calculations.

I hereby declare under penalty of perjury that the foregoing information is true to the best of my knowledge and belief.

S/William A. Hatleman
William A. Halteman, Ph.D.

WILLIAM A. HALTEMAN
STATISTICAL CONSULTANT
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October 4, 1995

Brent Singer, Esq.
Rudman & Winchell
Bangor, ME 04401

Dear Mr. Singer:

The problems you posed to me can all be set into the following context:

Suppose an event has two and only two possible results. (These results are usually denoted as "success" for the result of interest and as "failure" for the other.) Suppose, further, that the probability (or chance) that a "success" occurs on any particular event is denoted by *p*. (Necessarily, then, the probability that a "failure" occurs is given as *(1-p)*.) The chance that a "success" never occurs in some number, say *n*, independent repetitions of the event is given by:

$$(1-p)^n$$

An immediate consequence of this result is that the chance a "success" ever occurs (i.e., one or more times) in the n repetitions is given by:

$$(1 - (1 - p)^n$$

These results can be used to solve three problems:

a) Given *p* and *n*, one may compute the chance that a "success" never occurs.

b) Given p and a specified limit for the probability that a "success" never occurs, one may compute the minimum number, n, of repetitions of the experiment to achieve or exceed that limit.

c) Given n and a limit for the probability that a "success" never occurs, one may compute the probability, p, that a "success" occurs on any trial.

The problems you have stated fall into category b. If the specified limit that a success ever occurs is denoted by L, the pertinent equation becomes:

$$L = 1 - (1 - p)^n$$

After some rearrangement, solution for n is obtained from:

$$n = \frac{\log(1 - L)}{\log(1 - p)}$$

This is the formula I used to obtain the results presented in the declaration.

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IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MAINE

SIDNEY ABBOTT, Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D. Defendant	]	

Declaration of Donald Wayne Marianos, D.D.S., M.P.H.

Pursuant to 28 U.S.C. § 1746, I, Donald Wayne Marianos, D.D.S., M.P.H., declare that:

I have read Defendant's Memorandum of Law in Opposition to Plaintiff's Motion for Summary Judgment, and the Second Declaration of Sanford F. Kuvin, M.D. Based upon my education, experience and expertise I offer the following additional opinions in this case:

1. While statistical modeling, such as that employed by Dr. Kuvin, may be useful in estimating orders of magnitude of the risk of HIV transmission from an HIV-infected patient to dentist, these models are based on a multitude of assumptions. Estimates derived from theoretical modeling can only be as accurate as the sum of the model's individual elements.

2. Dr. Kuvin uses an estimate of 1.5 needlestick injuries per 100 injections given by dentists. In a more recent study (Cleveland, JADA 1995) in which dentists were observed for 6 months, zero injuries were recorded during over 16,000

injections. Siew's prospective "Diary" study (JADA 1995) found an annualized mean injury rate of about one needlestick every two years.

3. Dr. Kuvin uses the estimate that 3 out of 1000 accidental needlesticks from HIV-contaminated needles will result in occupational acquisition of HIV. While this is an accurate statistic, it represents the risk to all health care workers generally, not dentists specifically. The risk of HIV transmission following a percutaneous exposure to HIV infected blood is likely to depend on several factors, including the size of inoculum (i.e., the amount of blood injected). Because the size of anesthetic needles used in dentistry is much smaller than that used in most medical settings, the majority of needlesticks among dental workers will involve smaller, rather than larger amounts of HIV infected blood. It is the position of the CDC that the risk of acquiring HIV occupationally from a needlestick in the dental setting is so low that it cannot be accurately quantified.

4. Dr. Kuvin also uses the assumption that 1 out of 3 needlestick injuries to dentists results in parenteral exposure to patient blood. I am unaware of any study supporting this assumption.

5. Dr. Kuvin is incorrect when he states "1/3 of all reported exposures for dental workers have HIV, and no other identifiable risk factors except for occupational exposures." Dr. Kuvin has confused CDC's prospective needlestick study (described in the Gooch article attached to Dr. Kuvin's affidavit) and CDC surveillance system for HIV/AIDS. The nineteen dental workers described in the Gooch article were voluntarily enrolled in a national prospective study of health care workers exposed to HIV infected blood. To our knowledge, none of these exposures resulted in HIV transmission to an enrolled dental worker.

This study is completely separate from the CDC's ongoing surveillance system, in which six dental workers with AIDS have been identified as having no identifiable risk factors other than percutaneous or mucocutaneous exposure to blood. As noted in my previous affidavit, it is likely that a number of health care workers identified as having no identifiable risk factors actually had nonoccupational risk factors that either they or their proxies did not report or recognize.

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UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

SECOND AFFIDAVIT OF RANDON BRAGDON, D.M.D.

I, Randon Bragdon, D.M.D., being duly sworn, state as follows:

1. In his supplemental affidavit, paragraph 11, Ms. Abbott's expert witness John A. Molinari, Ph.D., made the following statement:

> According to Dr. Bragdon's Statement of Facts, his practice is to place his fingers in the patient's mouth next to the needle while he injects the anesthetic. The currently recommended procedure involves removing one's hand from the injection site, retracting the cheek with a mirror, and injecting anesthetic with the other hand. This method virtually eliminates any risk that a dentist will be stuck with a needle during the injection of a local anesthetic.

2. The recommendation made in this statement most likely comes from a person who has no experience giving intraoral injections. It does not surprise me that this novel but dangerous technique is not described in the book Practical Infection Control in Dentistry Dr. Molinari co-authored with James A. Cottone, D.M.D., M.S. and Geza T. Terezhalmy, D.D.S., M.A.

3. Dr. Molinari also failed to alert the audience to this novel technique during his speech at the American Conference on the Dental Management of HIV Disease held in Chicago June 16-

18, 1995. The title of his speech was "Infections of Risk to Healthcare Providers" and did include discussion of percutaneous injuries including needlesticks. Furthermore,

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another plaintiff's expert witness Dr. Donald Marianos gave a speech at the same meeting entitled "Update on Transmission Risks for Providers, Patients Needlestick, Splash Injury Implications, and Protocol for Healthcare Providers." Dr. Marianos also failed to alert the audience to this new technique for giving injections. Had he done so, I believe the audience of experienced professionals would have been incredulous.

4. Another plaintiff's expert witness, Dr. Deborah Greenspan, in the book AIDS and the Mouth she co-authored with Dr. John S. Greenspan, Dr. Morten Schiodt, and Dr. Jens J. Pindborg (deceased) makes no mention of such a technique. In fact, Dr. Greenspan states on pages 180 and 181: "In 1987, an ADA survey reported that general dentists gave an average of 177 local anesthetic injections per month and that the rate for needlestick injuries was 1.5 per 100 injections."

5. Also attached is the program brochure for the three day conference entitled "Frontline Healthcare Workers: National Conference on Prevention of Sharps Injuries and Bloodborne Exposures" held in Atlanta on August 14-16, 1995. This conference did not discuss Dr. Molinari's novel solution for prevention of needlesticks either.

6. It is well-known that the vast majority of needlesticks which occur during dental injections are the result of unexpected movements of either the patient or someone else in or entering the operatory. Most people do not realize that the reason that most needlesticks occur in the mouth is that it is the lesser of two evils. When control of a needle is lost either in or approaching the mouth by an unexpected movement, the patient must be protected from potentially serious injury. Often, the only practical way of preventing injury to the patient is for the dentist to position his or her hand and fingers so as to steady the head and jaw and to

block an errant needle. Better the dentist be stuck than the patient seriously injured.

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DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

Centers for Disease Control
and Prevention (CDC)
Atlanta GA 30333

Oct 30 1995

The Honorable Daniel P. Moynihan
United States Senate
Washington, D.C. 20510-3201

Dear Senator Moynihan:

I am responding to your letter on behalf of Dr. Paul Dworkis regarding his concerns about the efficacy of barrier precautions, such as gloves and masks, to prevent the transmission of bloodborne organisms during the provision of dental care.

The Centers for Disease Control and Prevention (CDC) published Recommended Infection Control Practices for Dentistry in 1986 and updated them in 1993. CDC infection control recommendations are intended to address a broad spectrum of dental procedures and are developed and updated with advice from outside consultants, including experts in infection control and public health. While many infection control recommendations are based on extensive epidemiologic data, others are based on established principles of infection control.

Gloves and masks (also known as personal protective equipment) are worn to provide a protective barrier and to prevent gross contamination of skin and mucous membranes of health care workers when touching blood or saliva contaminated with blood. The efficacy of these precautions (combined with other measures, including handwashing after removing gloves) to reduce blood contact with skin and mucous membranes has been well documented in the scientific literature.

Enclosed are references that support the use of personal protective equipment during medical and dental procedures. In addition,

enclosed are the two latest articles that establish the efficacy of such practices.

I hope this information is helpful.

Sincerely,
s/David Satcher
David Satcher, M.D., Ph.D.
Director

Enclosures

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Are Universal Precautions Effective in Reducing the Number of Occupational Exposures Among Health Care Workers?

A Prospective Study of Physicians on a Medical Service

Edward S. Wong, M.D., Genner L. Stotka, MD, Vernon M. Chinchilli, PhD, Denise S. Williams, MD, O. Gen Stuart, MD, Shegan M. Markowitz, MD

Using a daily questionnaire, we prospectively studied 277 physicians from two hospital medical services for incidents of exposure to blood and body fluids and barrier use before and after the implementation of universal precautions. We found that implementation significantly increased the frequency of barrier use during exposure incidents from 54% before implementation to 73% after implementation of universal precautions. Implementation led to a decrease in the number of exposure incidents that resulted in direct contact with blood and body fluids (actual exposures), from 5.07 to 2.66 exposures per physician per patient care month, and to an increase in averted exposures in which direct contact was prevented by the use of barrier devices, from 3.41 exposures per patient care month before implementation to 5.90 exposures per patient care month after implementation. Implementation affected neither the types of body fluid or procedures involved nor the overall rate of exposure incidents (8.5 per patient care month) but, through an increase in barrier use, it *did* prevent direct contact with blood and body fluids and thus converted what would have been an actual exposure into an averted one. We conclude that universal precautions were effective in reducing the risk of occupational exposures among physicians on a medical service.

(JAMA. 1991:265:1123-1128)

HEALTH CARE workers are at risk for acquiring hepatitis B virus (HBV) and human immunodeficiency virus type 1 (HIV-1) infections through occupational exposures to blood and certain body fluids (BBF). Exposure to HIV-1 is particularly worrisome because of the high mortality rate and the current lack of a curative treatment. Recent studies have shown that the risk of acquiring HIV-1 infection is approximately 0.4% following percutaneous exposure and even less for mucous membrane and cutaneous exposures. These results, while reassuring, should be interpreted

with caution since the overall risk to the health care worker is dependent both on the rate of transmission per episode of exposure and the cumulative number of exposures sustained over time. Few studies have attempted to determine the cumulative risk prospectively, relying instead on periodic surveys that ask health care workers to recall exposures sustained over prolonged intervals. Such studies are subject to the biases inherent in retrospective methods.

In 1987, the Centers for Disease Control (CDC) recommended that all hospitals adopt an infection control policy of "universal precautions" (UPs). Under this policy, health care workers are to assume that the BBF of all patients are infected with blood-borne pathogens and that they should, therefore, protect themselves with barrier devices when anticipating contact. These precautions are expensive; one recent study estimates that they cost at least $336 million per year in the United States to implement and maintain. While theoretically useful, there is currently no evidence that UPs will actually reduce the number of exposures sustained by health care workers. The possibility exists that UPs may even increase certain kinds of exposures; for example, the use of gloves may interfere with tactile input and increase the number of injuries with sharp instruments during procedures. In addition, if previous studies on compliance with infection control policies hold true, health care workers may not adhere to suggested precautions.

In order to assess the hazards of occupational exposures and the efficacy of UPs more accurately, we prospectively studied, with the use of a daily questionnaire, the frequency and types of exposures incurred by physicians during their care of patients on acute care medical wards before and after hospital-wide implementation of UPs.

SUBJECTS AND METHODS

Study Population and Design

Three acute care medical wards at the Hunter Holmes McGuire Department of Veterans Affairs Medical Center and

the Medical College of Virginia Hospital (both located in Richmond, VA) were chosen for study. The Veterans Affairs Hospital (VAH) has 814 beds and draws patients from the mid- and south-Atlantic region. The University Hospital (UH) is a 900-bed, urban, inner-city, tertiary care institution and a referral center for central Virginia. Ward A at the UH has 28 beds, while wards B and C at the VAH have 40 beds each. The monthly census for ward A and the combined monthly census for wards B and C during the study period averaged 105 and 192 patients, respectively. Patients on each ward were the primary responsibility of a house staff team that consisted of an attending physician, one resident, two interns, and two or three medical students. Neither institution has a ward for patients with acquired immunodeficiency syndrome; rather, ward assignments were made based on availability of beds.

The study population consisted of attending physicians, house staff, and medical students who rotated monthly through the study wards at both hospitals. From May 1, 1988, to January 31, 1989, these physicians were monitored prospectively for occupational exposures by means of a daily questionnaire. Participants were informed that the purpose of the study was to evaluate the frequency of occupational exposures to BBFs. They were not told that compliance with use of barrier devices was being monitored. We made no attempts to influence the use of protective devices or encourage excessive precautions. We requested names on the questionnaires but, once study participation was recorded (and during the third and fifth month, data verified), physicians were assigned a study number and personal identifiers were removed. Physicians were informed of this process to encourage their participation.

Questionnaires were passed out by a member of the ward team during attending rounds. We asked physicians to complete one questionnaire for each exposure incident sustained in the preceding 24 hours. An *exposure incident* was defined on the questionnaire as an occurrence in which the physician was exposed to the BBFs of a patient, regardless of whether the exposure resulted in direct contact with BBFs or not because of the use of barrier devices (direct contact avoided). The questionnaire sought the following information for each exposure: the date and time of

exposure, the procedure involved, the BBFs encountered, the contact site, and whether there was a cut, open wound, or inflamed skin at the exposure site. If the exposure site involved skin, physicians were asked to provide a semiquantitative assessment of the size of exposure; large (contact area the size of a hand), small (contact area the size of a quarter or less), or moderate (contact area intermediate between large and small). Physicians were also asked to specify which protective devices (gloves, gowns, masks, or goggles) they were wearing at the time of exposure; if they were wearing barrier devices; if the devices protected them from direct contact with BBFs; or, if they did not, would they have been protected had they donned barrier devices. Physicians experiencing BBF exposure on a given day were asked to check the "no exposure" response to calculate the denominator number of patient care days at risk).

Forms were collected daily. To determine compliance with the study, work schedules for physicians were obtained. Study compliance was calculated as the number of questionnaires returned divided by the number of days worked. Multiple responses by the same physician in 1 day (signifying multiple exposures during the same day) were counted as one response so as not to overestimate compliance. During the third and fifth month of the study, the information in every fourth questionnaire was verified by telephone or direct interview by one of the investigators within 24 hours of collection. The concordance between the results of the questionnaire and interview was 93%.

Universal precautions refers to the infection control policy by which the health care worker assumes that BBFs of all patients are infected with a bloodborne pathogen, advocates the use of gloves when touching mucous membranes and nonintact skin of all patients, and recommends the use of other appropriate barrier devices when indicated (eg. Masks, eye coverings, and gowns when droplets or splashes are likely to occur). Under UPs, the category of BBF isolation was eliminated, but other categories (eg. respiratory isolation) were retained. Body fluids included the following types: blood, wound drainage, cerebrospinal fluid, synovial fluid, pleural fluid, peritoneal fluid, and pericardial fluid, but not urine, saliva, feces, sputum, vomitus, or tears unless they contained visible blood.

All exposure incidents reported by physicians on the questionnaires were reviewed and classified as either (1) an actual exposure when direct contact with BBFs occurred because (*a*) barrier devices were not used or (*b*) when used, they failed to protect the health care worker (eg. A needlestick injury through a gloved hand); or (2) an averted exposure because barrier devices used by the physician at the time of exposure prevented direct contact with BBFs. Each exposure was further classified into one of the following four types (1) cutaneous exposure when the skin at the site of contact or underneath the barrier device in an averted exposure was intact; (2) nonintact skin exposure when the skin at the site of contact or underneath the barrier device in an averted exposure was inflamed or had a cut or open wound; (3) mucous membrane exposure at the eye or mouth; and (4) needlestick injury.

The study was approved by our institutional review board. Written informed consent of physicians was not required by our institutional review board because participation was voluntary and responses to questionnaires were anonymous.

Implementation of Universal Precautions

Universal precautions were not adopted as the official hospital policy until November 1, 1988. During the preimplementation period, both hospitals practiced the traditional category specific system of isolation by which isolation precautions were initiated in response to a patient's suspected communicable disease or diagnosis. Routine serologic screening for HIV-1 and HBV infections was not done at either hospital.

The implementation process began on September 26, 1988. A memorandum was sent to all health care workers defining the UP policy and listing a schedule of mandatory education sessions to review the new policy. The memorandum clearly stated that these sessions would be mandated by the Occupational Safety and Health Administration and that attendance was required.

At the UH, 40 separate educational training programs were given throughout October 1988. These sessions were presented by the hospital Epidemiology Unit and given in the form of a slide presentation. These 1-hour sessions reviewed the following: the etiology agents (HIV-1 and HBV), the modes of transmission, the

natural history of infection, and the risk of nosocomial acquisition. Universal precautions policy was explained, including the appropriate use of all barrier devices, the appropriate waste disposal procedures, and the proper use of cleaning agents to inactivate HIV-1. Proper handling of sharp instruments and needles was emphasized, and the correct procedure to report needlestick injuries was reviewed. Physicians received additional education on UPs through medical grand rounds and other teaching conferences

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in November 1988.

Barrier devices required for isolation precautions under the category-specific system of isolation were supplied by means of isolation carts placed outside patients' rooms. Under the UP policy, individual isolation carts were replaced by a centralized cart stocked with gloves, masks, gowns, and goggles. These carts, one per ward, were restocked daily. Each patient's room was supplied with a box of examination gloves and a puncture-resistant, leak-proof needle disposal unit. Every nurse was provided with a mouth shield to use for mouth-to-mouth resuscitation. Signs announcing the new policy of UPs were mounted strategically in the centralized nurses' station on each ward.

Implementation of UPs at the VAH followed similar programs and procedures with minor differences. The October training sessions were presented in the form of a 17-minute videotape and a 15-minute didactic lecture. Health care workers were given a multiple-choice examination before and after these sessions. Besides the initial September memorandum, the hospital Epidemiology Unit distributed a follow-up memorandum on UPs to all health care workers. A VAH medical grand rounds on UPs was given on September 30, 1988.

Statistical Analysis

The major outcome (dependent) variable of interest was the binary response relevant to whether the physician experienced either an actual or averted exposure when exposed to the patient's

BBFs. Since the same physicians were repeatedly sampled over time (both before and after implementation of UPs), it could not be assumed that the binary responses for an individual were independent. Therefore, the method of analysis proposed by Prentice was chosen. This approach can be considered a version of multiple regression in which the response is binary instead of normally distributed. Unlike logistic regression, the Prentice method of regression allows for correlation among repeated measurements on individuals and assumes no particular distribution.

Using the Prentice method, the following (independent) variables were evaluated for their effect on the outcome of barrier use; implementation of UPs, patient admissions to study wards, the type of hospital (VAH or UH); the number of exposure incidents sustained by physicians, the compliance rate of the physician in filling out study questionnaires (the number of questionnaires returned divided by the number of patient care days worked), the time of day when exposure occurred (day, evening, or midnight shift), and emergency status of the procedure associated with the exposure. The effect of implementation on two other outcomes (the number of actual and averted exposures) was also evaluated using the Prentice method. In the regression analyses, only data from the preimplementation period (May 1, 1988, to September 15, 1988) and the postimplementation period (November 16, 1988, to January 31, 1989) were used, omitting data from the transitional period when UPs were being implemented (September 16, 1988, to November 15, 1988). Results of our regression analysis of dichotomous variables were expressed as odds ratios (Ors) with 95% confidence intervals (Cis), calculated directly from the regression coefficients. With needlestick exposures, in which the incidence was very low and the regression approach was not applicable, a Mantel-Haenszel "test" was applied.

Data storage and processing were performed via the CLINFO program (BBN Software Products Corporation, Cambridge, Mass). Regression analyses were performed on a VAX 8650 with a program for Prentice's method of binary regression written by one of the investigators (V.M.C.) in PROC IML of SAS. The Mantel-Haenszel tests were performed via PROC FREQ of SAS.

RESULTS

The Epidemiology of Exposure Incidents

Of 294 physicians, who rotated through the three study wards during the 9-month study period, 277 (94%) physicians participated in our study. These physicians returned 4573 questionnaires during 6697 patient care days on the study wards. The compliance rate of the participating physicians for returning a questionnaire, after adjustment for multiple exposures experienced on the same day, was 67%. The monthly compliance rate ranged from 65% to 70% and there was no significant difference in compliance between months either before or after implementation of UPs.

A total of 1553 exposure incidents (actual and averted exposures) to BBFs were reported during the 9-month study period. Ninety-two percent of these exposures involved blood, either by contact (1379 exposures) or by needlestick injury (49 exposures). There were 25 exposures (1.6%) each to peritoneal and cerebrospinal fluid and 20 exposures (1.3%) to wound drainage. Exposures to all other body fluids were infrequent (Table 1).

[Table 1 and Table 2 omitted]

The site of exposure was the hand in 1545 incidents (99%); this included all 49 needlestick injuries. Physicians reported only two mucous membrane exposures (one involving the eye and one the mouth). Six exposures occurred on the face, but did not involve the eyes or mouth. Of 1496 cutaneous exposures, 76 (5%) occurred on nonintact skin (dermatitis or cut). The area of contact in cutaneous exposures was described as small or moderate in size 93% of the time. However, in 104 incidents (7%), the contact site covered an area the size of a hand or larger.

The procedures leading to exposure incidents are listed in Table 2. Eighty-nine percent of these exposures occurred during procedures that involve the insertion or manipulation of needles or catheters (719 exposures during venipuncture, 533 exposures associated with intravenous catheters, and 123 exposures during arterial punctures). Noninvasive procedures (eg respiratory

suctioning, wound care, or cleaning the patient) accounted for less than 3% of the exposures.

The Impact of Universal Precautions

Before hospital-wide implementation of UPs, the frequency of barrier use (gloves, gowns, or masks) by physicians during an exposure incident was 54%. The rate of actual exposures experienced by physicians during preimple-

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[Table 3 and Table 4 omitted]

mentation was 5.07 exposures per physician per patient care month (PCM), of which 4.23 were cutaneous exposures, 0.39 were needlestick injuries, and 0.45 were nonintact skin exposures. During this same period, physicians experienced averted exposures at a rate of 3.41 exposures per PCM. The majority of these exposures were cutaneous exposures (3.12 exposures), but averted nonintact skin exposures also occurred at a rate of 0.29 episodes (Table 3).

After implementation of UPS, the frequency of barrier use reported by physicians during exposures rose to 73%. The rate of actual exposures per physician after implementation fell to 2.66 per PCM, while the rate of averted exposures increased to 5.90 episodes per physician PCM (Table 3). The rate of needlestick injuries dropped from 0.39 to 0.15 exposures per PCM in the postimplementation period ($P = 123$, Mantel-Haenszel $_x^2$).

Comparison of data from the preimplementation and postimplementation periods by the Prentice method of regression revealed significant increases in the frequency of barrier use ($P = 4.7 \times 10$) and in the rate of averted exposures ($P - 2.0 \times 10$) and a significant decrease in the rate of actual exposures ($P = 6.7 \times 10$).

The adjusted OR for the effect of UPs on barrier use was 3.3 (95% of CI, 2.0 to 5.2). Thus, physicians were more than three times as likely to take barrier precautions during an exposure incident after implementation than before. The OR for the effect of

UPs on the probability of experiencing actual exposures was 0.3 (95% of CI, 0.2 to 0.5), an approximately threefold reduction in risk after implementation. The likelihood of avoiding an exposure through the use of barrier devices (averted exposures) increased threefold with the implementation of UPs (OR, 2.9;95% CI, 1.8 to 4.4).

Our analysis also revealed the following associations with barrier use, even after adjustment for the impact of implementation: physicians who were more compliant with the study questionnaire and those who suffered frequent exposures more often used barrier precautions, whereas, physicians performing emergency procedures used barrier precautions significantly less frequently than when performing non-emergency procedures. (Table 4).

The rate of exposure incidents (actual and averted exposures combined), the frequency distribution of procedures leading to exposure, the sites of involvement, and body fluids involved before and after implementation of UPs were compared and found not to have been affected by implementation.

COMMENT

In August 1987, the CDC published recommendations for the universal use of protective barrier devices to protect health care workers from blood-borne pathogens. However, despite the theoretical benefits of UPs and strong endorsements for their adoption, little evidence exists to show that UPs will protect health care workers as intended. Our study is one of the few to demonstrate that UPs can reduce the number of occupational exposures. We demonstrated by prospective surveillance that implementation of UPs was associated with a significant reduction in the number of actual exposures sustained by physicians from 5.07 exposures per PCM in the preimplementation period to 2.66 exposures per PCM in the postimplementation period. We attribute this benefit largely to the effect of implementation on barrier use, since the decrease in actual exposures was inversely proportional to the increase in the frequency of barrier use and to the increase in the number of averted exposures (Table 3). Although we cannot be certain that implementation was the only factor, we are unaware of any other factor or change in patient care routine occurring during the study period that may have contributed to this reduction. In

particular, we consider the number of patients admitted to study wards to be an important and potentially confounding variable since admissions directly affect the number of diagnostic procedures and patient care manipulations performed and, therefore, exposure risk. However, the monthly census was similar in both the preimplementation and postimplementation periods, and regression analysis found it not to be a contributing factor (Table 4).

To the extent that implementation reduced the number of actual exposures it should also reduce the risk of occupationally acquired blood-borne infections. Variables that affect acquisition include the cumulative number of exposures, the transmission rate per exposure, and the prevalence of disease. For HIV infection, needlestick injuries carry the highest risk of transmission at 0.4% per episode of exposure. Since the rate of needlestick injuries fell from 0.39 to 0.15 episodes per PCM (a reduction of 62%) we estimate that the implementation of UPs should reduce the risk of occupational HIV infection by the same rate (62%) since the other variables (transmission rate and prevalence) remain unchanged. For HBV, implementation of UPs should have an even greater impact since in most localities the prevalence of HBV is higher than HIV, the efficacy of transmission per exposure episode is greater (6% to 32% per

exposure episode vs 0.4% for HIV), and exposures other than needlestick injuries (mucous membrane or nonintact skin exposures) have been known to transmit infection.

We noted in our study that the majority (99%) of the exposure incidents involved the hand, with only eight exposures affecting the face or mucous membranes. In addition, in 345 (89%) of 389 actual exposures when barriers were not used, physicians reported that, had gloves been worn, their exposures could have been avoided. We also noted that the number of needlestick injuries fell from 0.39 episodes per month on the study wards before implementation to 0.15 episodes per month after implementation of UPs, a trend that was also observed in another recent study. A possible explanation for this reduction might have

been improved access to disposal units (one provided per patient room), which, although unlikely to affect the frequency of needlestick injuries associated with procedures, could have reduced the number of needlestick injuries related to transport and inappropriate disposal. The CDC recommendations emphasized all elements of UPs equally. Our findings suggest that the use of gloves is the largest contributor to the efficacy of UPs.

We were also able to identify problem areas that led to occupational exposures (Table 2); this information should be useful in efforts to further reduce such exposures. Specifically, we noted that venipuncture and the insertion or manipulation of intravenous access catheters led to 1252 (81%) of 1553 of reported exposure incidents among physicians. This suggests that, at a minimum, efforts should be directed specifically to improve physicians' techniques in venipuncture and catheter insertion and care. Alternatives include the use of phlebotomy and intravenous care teams. Although the value of such teams remains controversial, we noted that the proportion of exposure incidents related to catheter insertion and maintenance was significantly lower at the UH, which had an intravenous care team, than at the VAH, which did not (64 of 290 vs 469 of 1263, $P = .001$, $_{x^2}$).

Our study design raises several methodologic issues requiring comment. First, we did not begin our study until 8 months after the CDC published their recommendations for UPs and, as a result, many of our physicians were already practicing barrier precautions before official hospital adoption of the policy. Had we begun our study earlier, the baseline frequency of barrier use would have been lower and the impact of UPs might have been more dramatic. Despite this, we were still able to demonstrate a substantial gain in the frequency of barrier use and a reduction in occupational exposures. Second, we relied on a daily questionnaire as our surveillance tool. We would have preferred direct observation, but to adequately monitor several hundred health care workers over the extended study period would have required a large number of observers that, even if practical, would have likely introduced its own form of bias through the observer or Hawthorne effect. In addition, the process of completing questionnaires itself can have an effect on the use of barrier devices by focusing attention on occupational exposures. However, since the same questionnaires were used before and after implementation of UPs,

whatever effect it may have should be qualitatively and quantitatively similar before and after implementation, leaving the difference in barrier use between these two periods--the outcome of interest--unchanged.

Third, the participation rate among physicians in filling out the questionnaires was 67%. This rate is less than ideal, but perhaps should not be unexpected given voluntary participation and patient care duties of a busy medical staff. Nonetheless, potential biases can arise. For example, we observed that physicians who were more compliant in filling out the questionnaires were also more likely to use barrier devices (Table 4); thus, any improvement in participation during the postimplementation period could theoretically account for the improved barrier use. However, as noted, we observed no significant variation in the participation rate from month to month. We should emphasized that, although compliance with questionnaires was 67%, study compliance, when judged by the number of physicians who participated out of the pool of physicians who rotated through the study wards, was 94%. Thus, there was little selection bias in the types of physicians who participated.

Finally, our questionnaire solicited data on the frequency of barrier use during procedures and patient care manipulations associated with an exposure incident. Thus, barrier use during procedures not leading to an exposure to BBFs could not be ascertained. But, because a physician's decision on whether to don barrier devices is made before an exposure incident has occurred, we believe that our data, although based on a subset, can validly be extrapolated as representative of overall usage patterns.

The strength of our study lies in its prospective design, which allowed us to sample our cohort of physicians daily, obtaining detailed information on the epidemiology of exposures as well as on their incidence before and after the implementation of UPs. The validity of our results has recently been corroborated by Lynch et al. Based on periods of direct observation, these authors found overall glove use among their health care workers to be 61% before implementation and 81% after implementation of body substance isolation, a system of infection control similar to UPs. Thus, their results are remarkably similar to ours despite differences in method. Unlike their study, however, our investigation was extended to assess what impact the increase in

barrier use had on the incidence of occupational exposures, the critical issue.

Based on our findings, we conclude that UPs, as advocated by the CDC, are effective in protecting physicians from occupational exposures to BBFs. We should emphasize that our study was conducted at two urban medical centers. Procedural skills of health care workers, the frequency of performance of invasive vs. noninvasive procedures, and the prevalence of infections (HIV-1 and HBV) all affect exposure risk and these variables will differ from service to service as well as from institution to institution. For example, Gerberding et al recently reported on the risk of occupational exposures among operating room personnel. The differences in the epidemiology and incidence of exposures between their study and ours highlight differences in exposure risk between medical and surgical personnel. Whether and to what degree varying exposure risk affects the efficacy of UPs is an important issue that warrants further investigation. In addition, more prolonged follow-up studies will be needed to evaluate how long the effect of implementation persists and the need, if any, for further intervention to maintain compliance with UPs.

CLINICAL STUDIES

Frequency of Nonparenteral Occupational Exposures to Blood and Body Fluids Before and After Universal Precautions Training

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PURPOSE: During annual periods before and after Universal Precautions training, we compared the frequency of health care workers' self-reported cutaneous exposures to blood and various body substances from any patient and from patients presumed infected with human immunodeficiency virus type 1 (HIV-1).

SUBJECTS AND METHODS: Self-reported cutaneous exposures to blood, sputum, urine, feces, and other body substances were evaluated separately in 559 workers during the first survey and 269 workers during the second.

RESULTS: Mean annual blood exposures decreased from 35.8 to 18.1, and mean annual exposures to all substances decreased from 77.8 to 40.0 ($p < 0.001$ for both determinations).

Two matched analyses of a subset of 200 participants who completed both surveys had similar results. Reported exposures to blood, presumably infectious blood, sputum, presumably infectious sputum, and urine were significantly decreased. Participants were tested for antibodies to HIV-1; no participant reporting cutaneous exposures acquired HIV-1 infection. The upper bound for the 95% confidence interval for the risk of HIV-1 infection associated with a single cutaneous exposure was 0.04% for blood presumed to contain HIV-1 and 0.02% for any body substance presumed to contain HIV-1.

CONCLUSIONS: These data suggest that Universal Precautions training significantly decreased but did not eliminate cutaneous exposures to blood and body substances. The results further suggest that the risk for HIV-1 infection associated with cutaneous exposures is substantially lower than the risk associated with parenteral exposures.

Health care worker concern regarding risks for occupational acquisition of the human immunodeficiency virus type 1 (HIV-1) and hepatitis B virus (HBV) has contributed to the widespread implementation of a set of United States Public Health Service recommendations, frequently referred to as "Universal Precautions" [1]. Whereas these guidelines are designed to minimize health care worker exposures to blood and body substances, epidemiologic studies designed to assess workplace exposures to blood and body substances containing HIV-1 are limited. Available data have been obtained primarily from anecdotal reports and studies focusing on health care workers who have sustained parenteral injuries [2]. In aggregate, these studies have provided a reasonably precise estimate of the magnitude of risk for acquiring occupational HIV-1 infection as a result of a parenteral exposure (0.4% per exposure event) [3], but have offered only limited insight into the epidemiology and risks for HIV-1 infection associated with other types of occupational exposures.

Even though the epidemiology of mucous membrane and cutaneous exposures to blood, semen, vaginal secretions, and other body substances in the health care setting has not been well studied, transmission of HBV by these routes has been documented [4]. The Centers for Disease Control (CDC) estimates that approximately 12,000 health-care workers will develop serologic evidence of occupationally acquired HBV infection each year [5]. Extrapolating from data provided by Fedson [6] and Hoofnagle [7], of the 12,000 exposed, fewer than half will recall discrete occupational exposures.

In contrast, transmission of HIV-1 by nonparenteral routes has been documented infrequently and only anecdotally [8-11]. In May 1987, CDC reported three occupational HIV-1 infections that apparently resulted from nonparenteral exposures to blood containing HIV-1 [12]. Motivated both by this report and by the relative paucity of epidemio-

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logic information about nonparenteral exposures and their attendant risks in the health care setting, we developed and distributed a questionnaire to assess the frequency of cutaneous exposures to patients' blood and body substances among health care workers working at the clinical Center. National Institutes of Health (NIH). Subsequently, CDC issued guidelines for implementation of Universal precautions, and these policies were aggressively introduced into our hospital. The study reported herein had two objectives:

1. Assess and compare the self-reported frequencies of cutaneous exposures to blood and body substances among health care providers during a 12-month period before and a 12-month period subsequent to the implementation of Universal Precautions; and

2. Estimate the upper bound of the 95% confidence interval for the risk of occupational transmission of HIV-1 associated with nonparenteral exposures.

SUBJECTS AND METHODS

Since 1983, we have prospectively assessed the risk for occupational transmission of HIV-1 in a cohort of health care workers providing clinical care for the more than 1,500 HIV-1-infected patients at the Clinical Center. After providing informed consent, participants complete a confidential questionnaire and provide at least annual blood samples for serologic testing. Participants are identified only by a study number [13]. To gather information on non-parenteral exposures, study participants were asked to complete two supplemental confidential questionnaires.

The questionnaires were designed and pretested in June 1987. The initial survey was distributed during August 1987, before Universal Precautions policy implementation; the 12-month reference period of this first survey was from August 1, 1986, through July 31, 1987. A second identical questionnaire was distributed during April 1989, subsequent to Universal precautions implementation and employee training; this 12-month reference period was from April 1, 1988, through March 31, 1989.

At the time of distribution of the second survey, for logistical reasons, our health care worker study had been restructured to exclude individuals whose only opportunity for occupational exposures was in the research laboratory. For the second survey period, we directed surveys to those health care workers available for follow-up (i.e., not lost to staff turnover); hence, for the second survey, 285 fewer questionnaires were distributed.

The surveys requested that participants estimate separately the monthly occurrence of cutaneous exposures to patients' blood, sputum or oral secretions, urine, feces, and other body substances during the prior 1-year period. Participants were also asked to estimate average monthly cutaneous exposures to blood and body fluids presumed to have come from HIV-1-infected patients during the prior 1-year period. In most instances, participating staff were aware of patients' HIV-1 infection status either as a result of direct clinical interaction or by assumption of infection if the patient was placed into our hospital's isolation category "Blood/Body Fluids Precautions." The categoric responses provided in the questionnaires for the frequency of both types of exposures on a monthly basis were: 0, 1 to 5, 6 to 20, and more than 20.

Participants had at least annual serologic testing for anti-HIV-1 by a licensed commercial enzyme immunoassay (Dupont HIV ELISA, E.I. Dupont Nemours Laboratory, Wilmington, Delaware). Serologic data were linked by the participant's study identification number to exposure data reported on the questionnaires. These data were then used to provide an estimate of the upper bound of the 95% confidence interval for the risk of nosocomial transmission of HIV-1 associated with nonparenteral exposures.

Universal Precautions Policy Implementation and Training

The Clinical Center's Universal Precautions policy was impelemented in November 1987. Each hospital department reviewed and revised existing infection control policies, categorized tasks in accordance with the Occupational Safety and Health Administration's standards, and indicated which barriers were required for each task.

All hospital personnel whose jobs entailed potential exposure to patients' blood and body substances were required to attend a training session and complete a written examination. In addition to attending a formal training session, employees were exposed to information about Universal precautions from posters, memos, and newsletters distributed throughout the Clinical Center. New employees were required to attend a training session before reporting for active duty. Documentation of physician training in Universal Precautions was integrated into our hospital's credentialing process. More than 2,200 health care workers were trained, and we conservatively estimate that more than 95% of the study participants had received the training before administration of the second survey.

Access to protective barriers (gloves, masks, gowns, and eyewear) was facilitated by providing supply carts to all patient-care areas. "Code stations," consisting of gloves and pocket masks with

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[Table 1 omitted]

one-way valves to be used by cardiopulmonary resuscitation first responders, were installed throughout the hospital and in every patient's room.

Analytic Methods

We compared the total number of exposures reported for each body substance for the two 12-month periods. We calculated annual totals of exposures using the midpoint of the chosen range, and the lower limit of the "more than 20" category (i.e., 21). For example, if a participant selected "6 to 20" to indicate the number of monthly cutaneous exposures 13 x 12 = 156 was used to determine the annual number of exposures. We calculated the annual mean number of cutaneous exposures for each body substance by dividing the number of reported exposures for the 12-month survey period by the number of participants at risk for cutaneous exposure to blood and body substances. The annual mean numbers of exposures before and after Universal Precautions training were compared by the two-sample Wilcoxon test.

Responses from the subset of participants who completed both questionnaires were compared in two types of matched analyses. In the first, a one-sample (paired) Wilcoxon test was used to compare the mean numbers of each type of exposure. In the second matched analysis, for each body substance, each participant was cross-classified in a 4 x 4 frequency table, with the rows representing the four potential categoric responses on the first survey, and the columns representing the second survey. These tables were analyzed for symmetry and conditional symmetry [14]. Symmetry is a test for equal movement across the diagonal. The occurrence of symmetry would mean that any change occurring between the two surveys could be attributed to chance alone. Rejecting the hypothesis of symmetry implies that a statistically significant change has occurred between the surveys. One type of change is a constant proportion moving across the diagonal. Conditional symmetry is the test conducted to assess if this type of change occurred. If the hypothesis of conditional symmetry is not rejected, it is not possible to reject the claim that a significant and constant change has occurred. Chi-square statistics were calculated for each of the body substance tables, with six degrees of freedom for the symmetry case, and five degrees of freedom for the conditional symmetry case. In addition, the constant proportion in the conditional symmetry case was estimated.

We used the Poisson distribution to calculate the upper bound of the 95% confidence interval for the risk of HIV-1

infection associated with cutaneous exposure to blood or body substances presumed to contain HIV-1.

RESULTS

Table I displays the numbers of participants who received and who responded to questionnaires for both survey periods. The response rate was 67% for the first and 50% for the second survey. Work duties that placed respondents at risk for cutaneous exposure to blood or body substances were reported by 85% of participants in the first survey and 77% in the second survey. During the first study interval, 60% of the 559 health care workers at risk for cutaneous exposures reported at least one such exposure. Significantly fewer health care workers (51% of 269, $p < 0.05$, $_x^2$) reported cutaneous exposures during the second 12-month period. Whereas the percentage of respondents reporting at least one such exposure had decreased from the first survey to the second, cutaneous exposures to blood and body substances continued to occur frequently.

The numbers of cutaneous exposures to each body substance reported for the two study intervals are shown in **Table II**. During the first 12-month period, prior to Universal Precautions training, 309 (55%) of the 559 participants at risk for exposures estimated a yearly total of 20,028 cutaneous exposures to blood; the mean number of annual cutaneous exposures to blood per participant was nearly

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[Table II Omitted]

36. Respondents reported a mean range of from four to 15 cutaneous exposures to sputum, urine, feces, and other body fluids. Study participants self-reported an estimate of more than 43,000 total blood and body substance exposures for this time period. A mean number of 77.8 cutaneous exposures occurred annually per

participant during the year surveyed before Universal precautions training.

Results from the second survey, administered subsequent to Universal Precautions training, are also presented in Table II. Of the 269 participants at risk for exposure, 106 (39%) estimated a yearly total of 4,872 cutaneous exposures to blood. The mean number of annual cutaneous exposures to blood per respondent was 18.1. A range of from one to nearly 10 cutaneous exposures to sputum, urine, feces, and other body fluids was reported. The mean number of annual exposures per participant for all body substances was 40.0. Except for urine, which was nearly significant, and "other body fluids," the mean number of cutaneous exposures to each category of body substances, as well as the mean total exposures, decreased significantly compared with the first survey period (Table II).

During both study periods, all Clinical Center patients known to harbor bloodborne pathogens (e.g., HIV-1, HBV, non-A, non-B hepatitis, and human T-cell lymphotropic virus type 1) were routinely placed into the isolation category called "Blood/Body Fluids Precautions." The specifications for this isolation category are virtually identical to the guidelines published by CDC [15] and are very similar to Universal Precautions. Adherence to these precautions should have minimized, if not eliminated, cutaneous exposures to blood or body substances known to contain these pathogens. Despite the existence of the Clinical Center's "Blood/Body Fluids Precautions" policy, a large number of exposures to blood and body substances from patients presumed to be infected with HIV-1 was reported. The numbers of cutaneous exposures from this higher-risk patient population, categorized by body substance, are illustrated in **Table III.** During the first period, health care workers estimated 6,528 cutaneous exposures to blood from HIV-1-infected patients, for an annual mean number of almost 12 exposures per health care worker at risk. The mean numbers of annual exposures to sputum, urine, feces, and other body fluids from infected patients were each less than five per participant at risk. A total of 14,256 cutaneous exposures to all blood and body substances from patients presumed to be infected with HIV-1 was estimated, for a mean annual rate of 25.5 cutaneous exposures per participant at risk.

Cutaneous exposures to blood and body substances from patients presumed to be infected with HIV-1 continued to be reported during the second study interval. In every body substance category, the reported mean numbers of cutaneous exposures decreased from the first survey, although the decrease was not always statistically significant. A total of 1,428 cutaneous exposures to blood thought to contain HIV-1 and 3,096 cutaneous exposures to any body substances from infected patients was estimated by participants. The mean number of annual blood and body substance exposures from infected patients decreased significantly from 25.5 reported for the first study interval, to 11.5 exposures per participant for the second 12-month period <0.001).

Each participant was serologically tested for HIV-1 at least 6 months after his or her last self-reported cutaneous exposure. No HIV-1 infection occurred among participants reporting cutaneous exposures to blood or body substances presumed to have come from patients harboring HIV-1 during either survey period. The upper bound of the 95% confidence interval associated with cutaneous exposures to blood potentially containing HIV-1 is

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[Table III Omitted]

0.05% for the first survey and 0.21% for the second. The upper bound of the 95% confidence interval for the rate of HIV-1 transmission associated with a cutaneous exposure to any body substance believed to contain HIV-1 is 0.02% for the first annual period and 0.10% for the first annual period and 0.10% for the second annual period. If the two survey periods are combined, the upper bound of the 95% confidence interval for the rate of HIV-1 transmission associated with a cutaneous exposure to blood presumably containing HIV-1 is 0.04%. The upper bound of the 95% confidence interval for the rate of HIV-1 transmission

associated with a cutaneous exposure to a presumably infectious body substance is 0.02%.

Because the decrease of reported cutaneous exposures might be due to a different composition of health care workers in the two survey periods, we conducted an assessment of the subset of 249 participants who completed surveys for both time periods. Of the 249 who responded to both questionnaires, 200 reported work duties during both surveys that included handling, processing, and/or having direct contact with blood or body substances. Two matched analyses were performed on the data provided by these 200.

In the first analysis, one-sample (paired) Wilcoxon tests of the mean numbers of each type of body substance exposure were performed. Findings similar to those displayed in Tables II and III were identified in this subset. Of the 200 participants, during the first survey, 113 (56.5%) estimated a yearly total of 7,260 cutaneous exposures to blood. The mean number of annual cutaneous exposures to blood per eligible respondent was 36.3. Mean numbers of annual exposures to other substances ranged from 3.8 for feces to 18.2 for urine. The mean number of annual cutaneous exposures to all body substances per participant was 77.2.

During the second survey, only 80 (40%) of the matched participants reported a total of 3,456 exposures to blood, for a mean of 17.3 annual blood exposures. Mean numbers of annual exposures to other substances ranged from one for feces to 10 for urine. The mean number of annual exposures to all body substances was 40.0. This matched analysis demonstrated that the mean number of cutaneous exposures to each category of body substances, as well as the total number of exposures, decreased significantly from the first survey period.

For reported exposures to body substances from patients presumed to be infected with HIV-1, we performed a similar analysis. During the first survey, 52 (25.5) of the 200 matched participants reported 2,628 blood exposures (mean = 13.1 per participant at risk), compared with 23 (11.5%) participants who estimated 1,068 such exposures during the second survey (mean = 5.3 per participant at risk). This decrease in the mean number of blood exposures from presumed HIV-1 infectious patients was highly significant ($p < 0.001$). Similarly, during the first survey, the

mean number of annual cutaneous exposures to all body substances from infectious patients per participant at risk was 26.0; this mean decreased significantly to 11.3 per participant at risk during the second survey ($p < 0.001$).

An example of the second type of matched analysis for reported blood exposures is displayed in **Table IV**. This table demonstrates whether a particular health care worker's exposure frequency remained the same, increased, or decreased during the survey periods. The horizontal rows represent information from the first survey, the vertical columns represent information from the second survey. Table IV reveals that the numbers below the diagonal of symmetry (indicating the numbers of health care workers who reported decreased exposures from the first to the second survey) are much larger than the numbers above the diagonal (indicating the numbers of health care workers who reported increased exposures from the first to second survey). This suggests an overall decrease in cutaneous exposures. When the chi-square test for symmetry was performed, the null hypothesis of symmetry was rejected ($p < 0.001$). When the chi-square test for conditional symmetry was performed, the model of conditional symmetry was not rejected ($p = 0.27$). Hence, these data do not satisfy the statistical model of symmetry (the hypothesis of no change), but do not allow the rejection of the model of asymmetry (the hypothesis of constant change--see Subjects and Methods section). The reduction in cutaneous exposures to blood is statistically significant, and the proportion constant is 3.10. Any given individual in this group who showed a change between the two surveys was 3.10 times more likely to have had more exposures to blood during the first surveyed period than the second.

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[Table IV and Table V Omitted]

We conducted matched analysis for cutaneous exposures to other body substances in a similar manner, and **Table V** summarizes these results. Significance of proportional change (conditional symmetry), showing a decrease in reported exposures from the first to the second survey period, could be demonstrated for blood, blood from a patient presumed to be infected with HIV-1, sputum, sputum from a patient presumed to be infected with HIV-1, and urine. Although significance could not be demonstrated, a trend is shown for the other body substances that is similar to that demonstrated for blood, sputum, and urine.

COMMENTS

Despite data suggesting that so-called inapparent parenteral transmission is responsible for approximately 50% of occupationally-acquired HBV infections [6,7], health care workers' concern about the possibility of nonparenteral transmission of blood-borne infections was minimal until mid-1987. Cutaneous and even mucous membrane exposures seem to have been accepted as routine occurrences for clinical health care workers. In our own institution, for example, employees participating in a lon-

gitudinal study designed to measure the risk for occupational/nosocomial transmission of HIV-1 self-reported frequent nonparenteral exposures in study questionnaires, but only rarely formally reported such exposures to the NIH Occupational Medical Service [16].

The report of three instances of occupational/nosocomial HIV-1 infection following nonparenteral exposures to blood containing HIV-1 [12] resulted in increased interest in nonparenteral exposures in the health care setting. CDC responded to these increased concerns by assembling a panel of experts to evaluate the risks associated with all types of occupational exposures to blood and other potentially infectious body substances

and, ultimately, to make recommendations regarding the prevention of these exposures. The so-called Universal Precautions recommendations were a direct result of these deliberations [1]. These precautions were designed to minimize the risks for all kinds of occupational exposures to blood and other potentially infectious body substances in the health care setting.

Although our study provides some insight into the frequency of cutaneous exposures among health care workers at the Clinical Center, these results should be interpreted within the limitations of the study design. Survey participation was voluntary, and selection bias may have occurred. Participants' self-reported estimates of exposures were not verified by direct observation, and significant recall bias could occur in estimating the number of events during a 1-year period, especially for events that may occur commonly, such as skin exposure to blood. When respondents reported exposures to blood or body substances from patients presumed to be infected with HIV-1, knowledge of the patient's HIV-1 infection status could be variable. Source patient HIV-1 infection was not verified by serologic testing at the time of participants' self-reported cutaneous exposures unless the incident was formally reported to the Occupational Medical Service.

In order to minimize the potential bias of under-reporting cutaneous exposures for the second survey period, participants were not asked whether they had participated in Universal Precautions training. Although we could not be entirely certain of each participant's training status, more than 2,200 Clinical Center staff had received this training before the second survey was distributed. We concentrated training efforts on those personnel who had the highest likelihood of having contact with blood or other potentially infectious body substances, and we believe that more than 95% of the study participants had received the training before administration of the second survey.

The decrease in cutaneous exposures among health care workers in our hospital is most clearly demonstrated by the matched analysis of the 200 participants who responded to both surveys and whose work duties included the potential for contact with blood and other potentially infectious body fluids. Examination of data from these 200 health care workers suggests that the decrease in frequency of cutaneous exposures is not related to differences in the composition of the groups participating in both

study surveys (i.e., the difference was not due to "careless" exposure-prone health care workers being included in the first survey, but not in the second). Other possible sources of selection bias could overestimate the documented decrease in frequency of cutaneous exposures. For example, the 200 participants in our matched analysis could disproportionately represent health care workers who were more likely to respond to Universal Precautions training. Additionally, in the overall analysis, individuals who had lower frequencies of exposures could have been over-represented in the subset of those subjects who participated in the second, but not the first survey. To investigate this, we compared the 101 second survey respondents who participated in the second, but not the first survey, with the 249 second survey respondents who had also participated in the first survey. There were no differences in exposures of any blood or body fluid category (data not shown). Although our data do not directly address this latter issue, we believe that they suggest that this type of selection bias is unlikely.

Although well-designed and carefully constructed, the Universal precautions guidelines were not met with wholehearted enthusiasm. Several criticisms were leveled at these new infection control procedures, including, but not limited to: (1) the precautions are far too idealistic (i.e., in reality, it is impractical to try to treat all patients similarly); (2) there are no data suggesting that use of these extensive and expensive precautions is cost-effective; (3) the extensive use of barriers (gloves, gowns, masks, protective eyewear) would not have a substantial impact on the most important category of occupational exposure (i.e, that needlesticks and other "sharps" injuries are not likely to be prevented by using these barriers); (4) the exposures most likely to be prevented by using Universal Precautions (i.e., cutaneous and mucous membrane exposures) are associated with very low risks for transmission of blood-borne pathogens; and (5) there are no data suggesting that use of these precautions actually decreases occupational exposures.

With respect to the first of these criticisms (i.e., the impracticality of the recommendations), as a paradigm, the use of Universal Precautions for all

patients seems entirely rational. Nonetheless, several technical issues and human nature itself suggest that some flexibility in these guidelines may be appropriate [17]. From a practical standpoint, surgical colleagues argue that, historically, operative techniques have been varied in situations in which a patient is known to harbor a blood-borne pathogen. Further, surgeons argue that use of these labor-intensive and time-consuming techniques is not justifiable for all patients. Such precautions (e.g., no manual wound retraction, no hand-to-hand passes of sharp instruments, etc.) lengthen the time required for surgery, and, in so doing, increase the risk for other operative complications, including postoperative infection. Additionally, some health care workers argue that it is difficult to practice these precautions at the 100% level 100% of the time. One might postulate that a known or documented risk is more likely to be considered seriously, and managed appropriately, than a hypothetical risk. One possible explanation for the 50% reduction in health care workers' self-reporting occupational exposure to blood from HIV-1-infected patients in our study is that our educational efforts made them aware of the risks associated with handling HIV-1-contaminated blood, and that they modified their behaviors because of this known risk. Because of the manner in which our data were collected, our study provides only limited insight into the first criticism of the Universal Precautions recommendations.

An observational study conducted in an operating suite by Gerberding and her co-workers [18] provides at least a partial answer, as these investigators did not detect differences in the rates of adverse exposures experienced by operating room personnel in cases in which the patient was known to be HIV-1 infected when compared with cases when the patient was known to be HIV-1 seronegative.

Although implementation of Universal Precautions is expensive [19], questions about the cost-effectiveness of these precautions are difficult to address, since estimation of all of the costs associated with even a single occupational HIV-1 infection

(e.g., employee compensation, institutional liability, etc.) is difficult.

Whereas increased use of barriers is not likely to change the frequency of adverse occupational exposures to needles and other sharp objects in the health care setting, adherence to the other (i.e., the "nonbarrier") recommendations in the Universal Precautions guidelines may lower these exposures [1]. The Universal Precautions guidelines emphasize careful handling of needles and other sharp objects and include clear recommendations against recapping, bending, or clipping used needles [1]. These guidelines also stress techniques for appropriate disposal of used "sharps." Several studies, including the CDC Cooperative Needlestick Study, have estimated that more than 30% of occupational needlestick injuries would likely have been prevented if the Universal Precautions recommendations had been followed [20]. Conversely, some parenteral occupational exposures may be more difficult to avoid. Jagger *et al* [21] have suggested that some of these latter types of exposures may sometimes be related to problems with instrument or device design, while others are associated with improper use of certain medical devices.

Whereas on a "per-exposure" basis, nonparenteral exposures are likely to be associated with lower transmission rates than those associated with parenteral exposures, our study suggests nonparenteral exposures continue to occur quite frequently. In the absence of further behavior modification by health care workers, the frequency of these exposures, over time, may translate into a substantial workplace risk for some categories of health care workers.

On the basis of data from this study, we estimate 0.04% as the upper bound of the 95% confidence interval for the risk for transmission from a single cutaneous exposure to blood thought to have come from a patient infected with HIV-1. This risk is 10-fold lower than the risk associated with parenteral exposures to blood from HIV-1-infected patients (0.4%) [22]. Conversely, while 169 respondents to our surveys estimated they had 7,956 cutaneous exposures to blood thought to contain HIV-1 in the two 1-year study periods, only 37 parenteral exposures to blood containing HIV-1 were reported to the NIH Occupational Medical Service by all health care workers in the Clinical Center for the same periods. Such data may at least partially explain the observations of

Hoofnagle [7] and Fedson [6] that fewer than half of the health care workers who acquire occupational HBV infection recall a discrete exposure event.

With respect to the final cited criticism of the Universal Precautions recommendations (i.e., lack of evidence suggesting that Universal Precautions use is associated with decreased occupational exposures), data from our study document a decrease in the frequencies of self-reported cutaneous exposures to blood and other body fluids in our hospital in temporal association with the implementation of Universal Precautions. Although our data do not demonstrate a direct causal relation between training of our staff, the use of Universal Precautions, and the subsequent reduction in the number of cutaneous exposures, the data suggest that behavioral changes have occurred among health care providers

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in our hospital. We believe that the introduction, implementation, and maintenance of Universal Precautions in our hospital has contributed significantly to this reduction.

If cutaneous exposures are, in fact, responsible for a sizáble proportion of occupationally acquired bloodborne infections, and if, as we surmise, Universal Precautions education, training, and implementation resulted in the decrease in self-reported nonparenteral exposures seen in our study, the use of these precautions may ultimately significantly lower the occurrence of so-called inapparent parenteral transmission of blood-borne pathogens in the health care workplace.

[References Omitted]

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Journal of Medical Virology 33:47-50 (1991)

Human Immunodeficiency Virus-1 (HIV-1) in the Vapors of Surgical Power Instruments

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Cool vapors and aerosols produced by several common surgical power instruments and hot smoke plumes generated with electrocautery on known HIV-1 inoculated blood were gently bubbled through sterile viral culture media. Tissue culture cells were then added and cell infection was detected by the appearance of HIV-1 P-24 core antigen assayed by ELISA in the culture medium. HIV-1 was cultured from cool aerosols and vapors generated by a 30,000 RPM spinning router tip, an instrument similar to the Midas Rex and the Stryker oscillating bone saw. No infectious HIV-1 was detected in aerosols generated by a Valley Lab electrocautery or with a manual wound irrigation syringe known as a Travenol Uromatic irrigator. We have demonstrated that HIV-1 can remain viable in cool aerosols generated by certain surgical power tools and this raises the possibility of HIV transmission to medical personnel exposed to aerosols similarly generated during the care of HIV infected patients. Further work is required to determine whether such a risk exists but caution should be exercised by those exposed to aerosols generated during procedures on HIV-1 infected patients.

KEY WORDS: aerosols, surgical power tools, hot smoke plumes

INTRODUCTION

Surgeons and other health care workers are routinely exposed to significant and often prolonged aerosols generated by certain orthopedic and surgical procedures on bone, soft tissue, and

body fluids. When the tissues contain infectious agents, there is a possibility of transmission of the agent to personnel exposed to the aerosols. For example, papilloma virus DNA has been isolated from laser plumes from papilloma lesions, and nasal papillomas are seen to develop in the noses of physicians and operating room staff cumulatively exposed to laser/papilloma plumes [Garden et al., 1988].

Although HIV-1 appears to be transmitted most frequently by sexual contacts, and by percutaneous and perinatal exposures (Weiss et al., 1985: Melbye 1986. Henderson et al., 1986; Friedlan and Klein, 1986) there is a report of a laboratory worker possibly but not proven to be infected with HIV-1 by aerosols generated by ultracentrifugation or other manipulation of large quantities of cell culture medium containing tissue culture adapted HIV-1 strain HTLV III [Weiss et al., 1988; Barnes, 1988]. That case raises the possibility that individuals might be infected with HIV-1 when performing procedures which strongly aerosolize materials containing infectious HIV. The purpose of this study was to determine if infectious HIV-1 could be isolated from aerosols generated from human blood containing HIV-1 by common orthopedic and surgical procedures known to cause aerosols.

MATERIALS AND METHODS

Four milliliters of human banked O packed red blood cells known to be negative for CMV and HIV antibodies, 4 ml of sterile RPMI 40 with 10% fetal calf serum culture medium, and 1 ml of culture medium containing 10^5 tissue culture infectious doses (TCID) of HIV-1 (the HTLV III tissue culture adapted strain) were mixed. Such mixtures were individually subjected to electrocautery in the coagulation or the cutting mode, a high speed bone cutting router, an oscillating bone saw, and a wound irrigation syringe jet. Each of these procedures was conducted within a 2,400 mi Becton-Dickinson Vac-Rite operating room suction canister which was in a Biogard (Baker) laminer flow safety cabinet. The sampling-aspiration portal was positioned either 4 in (electrocautery experiments only) or 8 in (all others) above the source and consisted of a small 2 mm inner diameter plastic nipple from a 20 in K-50 Pharmaseal intravenous (IV) extension tube leading to the Leur-lok end of a 10 ml syringe containing 6-7 ml of sterile culture

medium. The cool aerosol or hot smoke plume generated by each procedure was suctioned from the canister via the i.v. extension tube through the

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sterile culture medium in the syringe at approximately 0.8 liter/min. MT-2 cells or human blood mononuclear cells (PBMC) of HIV-1 seronegative patients isolated by Ficoll-Hypaque density gradient centrifugation were then added to the culture medium to a final concentration of 5×10^5 cells per ml. The cells were placed at 37°C in a CO_2 incubator, fed biweekly, and culture medium was tested biweekly for 4 weeks by ELISA for P-24 HIV-1 core antigen (Abbott Laboratories) to assess whether the cells had been infected with HIV-1. For each culture experiment, culture medium containing HIV-1 and medium without virus were separately added to uninfected cells as positive and negative controls, respectively. A positive culture was defined as a P-24 ELISA value greater than 25 pg per ml (above the negative control background value) in two successive biweekly assays of culture medium.

In the electrocautery experiments, either 3 x 2 x 1 cm pieces of sterile thawed fresh frozen human skin and muscle samples or sterile filter paper were subjected to 1-4 min of cutting or coagulation while the HIV-1 containing blood was gently pipetted over the surface. The 2,400 ml canister filled with visible white smoke and the gaseous phase was aspirated for 1-4 min total time, resulting in clearing of the canister.

A cool aerosol was generated with a 30,000 RPM Black and Decker router with a 3.8 in morticing tip protruding 1.2 in inside the end of a 2,400 ml canister by slowly pipetting the HIV-1 containing blood directly into the horizontal spinning tip for period of 1-3 min. This created an immediate splattering around the base and lower third of the canister, while the 2 mm aspiration portal 8

in above the router tip was not visibly splattered. No liquid was observed in the inner lumen of the small tubing through which the gaseous phase was aspirated for 1-4 min.

The Stryker bone saw was used to generate an aerosol by pipetting three 9 ml aliquots of HIV-1 containing blood over the oscillating 1 x 3 in blade and then partially submerging the oscillating blade in the fluid for 1 min. Total times for aspiration of the gaseous phase were from 1 to 3 min.

The Uromatic irrigator, which is used in some emergency rooms to irrigate small lacerations, is a 10 ml syringe with a 20 gauge laminar flow soft plastic nozzle attached to a one-way valve which allows rapid refilling of the syringe from a reservoir and rapid lavage fluid ejection. Either 10 or 20 ejection repetitions (10 ml each) with the HIV-1 containing blood were performed and the overlying gaseous phase with no visible aerosol was aspirated for 2-3 min.

RESULTS

The results of all experiments are presented in Table I. Positive cultures for HIV-1 were obtained with cool aerosols generated by the effects of the high speed router tip and the oscillating bone saw on an HIV-1 containing blood mixture. The 30,000 RPM spinning router tip-generated aerosols were tested a total of 9 times under different time conditions. The aerosols were generated for 1-3 min then aspirated for a total of 1 to 4 min. A total of 5 aerosols were strongly positive for HIV-1 in cell culture with 480 to 642 pg/ml of P-24 HIV-1 antigen detected in culture medium. Four samples were negative. Two of these were the only two tested on PBMC which appeared to be less sensitive for this assay than MT-2 cells. One of the "negative" cultures had an initial P-24 Ag value of 111 pg/ml, but when this level was not sustained the aerosol was scored negative. The longest router and aspiration times (3 and 4 min) were all positive and the negative results occurred with the shortest times (1 or 2 min). One of the four Stryker saw generated aerosols was strongly positive (480 pg ml). Thus it appears that cool aerosols generated by these surgical power tools can contain infectious HIV-1.

Cool aerosols generated by the manual 10 ml syringe jet known as a Highland Irrigation device were negative for HIV-1 in

two experiments. Similarly, negative results were obtained with the Valley Lab Bovie set for maximal coagulation for times of 1,2, or 4 minutes in a total of 6 experiments and in 6 experiments with the instrument in the cutting mode. Half the runs were with HIV-1 containing blood on filter paper (used as a burn target in place of tissue) and half were with human skin and muscle specimens. In all of these experiments a visible aerosol or smoke was generated and this was suctioned through the test culture medium.

Four positive and three negative controls for the HIV-1 infectivity assay gave the expected results.

A separate experiment (performed by Don Jewitt et al., submitted) without virus demonstrated that blood aerosols of submicron and micron size were generated and collected by the procedures used in investigating HIV in aerosols. Aerosol particle size distributions measured with a quartz-crystal PC-2H cascade impactor (California Measurements, Inc.) were 0.28-14.0 μm for electrocautery.

DISCUSSION

These experiments show that infectious HIV-1 can be isolated from cool aerosols created from blood containing HIV-1 exposed to certain surgical power instruments such as a high speed spinning router (analogous to the Midas Rex) and the Stryker oscillating bone saw. Careful inspection revealed that no liquid or solid material passed through the small (2 mm) diameter 20 in length of tubing used to transfer the aerosol generated by these instruments within the canister to the test culture medium, indicating the virus retained sufficient infectivity in aerosols generated in this way to infect MT-2 cells in culture. Experiments with noninfected blood, utilizing an industrial quartz-crystal impacter, quantitated the large quantity of micron and submicron aerosols generated with each of our procedures.

The Uromatic syringe wound irrigator device, which

HIV-1 in Vapors of Surgical Power Instruments

[Table I Omitted]

can also be expected to generate cool aerosols less efficiently, did not result in positive cultures as strictly defined here, indicating that aerosols created in this way did not contain enough infectious HIV-1 to be detected in the assay used. The electrocautery in either coagulation or cutting mode created visible smoke plumes which were suctioned through culture medium, but no infectious HIV-1 could be detected, raising the possibility that high temperature of cautery may have inactivated HIV-1.

Although these experiments show that HIV-1 retains infectivity in aerosols generated in certain ways, they do not quantitate the risk of HIV transmission to personnel by such aerosols generated under clinical conditions, e.g., in the operating room. The failure to detect HIV-1 in aerosols generated by certain procedures (e.g., cautery or syringe jet) in our experiments does not exclude the possibility of clinical transmission by such procedures. Several variables could influence the risk of such transmission in a clinical setting. The concentrations of HIV in tissues and body fluids appear to vary widely in different infected patients and HIV is often cell associated. However, extracellular infectious HIV-1 in moderately high concentrations has been detected in plasma of at least some patients [Ernst et al., 1988]. The dose of the wild type HIV strains that might be necessary in an aerosol form to infect via respiratory or mucosal surfaces exposed to such aerosols, and whether such a dose can ever be generated in clinical situations are unanswered questions. Although our experiments raise the possibility that infectious HIV could be present aerosols generated in clinical practice, the large body of epidemiologic data on prevalence of HIV infections in different populations [Weiss et al., 1985; Melbye, 1986] would suggest that transmission by aerosols is not common. However, there are no published studies precisely examining medical or den-

tal personnel most heavily exposed to aerosols generated in care of HIV infected patients compared with appropriate controls, and such studies would seem desirable based on our findings. There are

studies showing that HBV seroconversion is higher in surgical and dental personnel even controlling for penetrating injuries, a result not inconsistent with an aerosol route of infection in some cases [Dienstag and Ryan, 1981]. An experimental approach that might provide some direct information about aerosol transmission of HIV would be exposure of chimpanzees to aerosols potentially containing HIV generated under conditions simulating different clinical procedures that create aerosols, or the use of some other model system such as simian immunodeficiency virus (SIV) and susceptible non-human primates for such experiments. Further work is also needed to better quantify the nature and size of the aerosol particles which can sustain infectious HIV and the length of time the virus remains viable in aerosol form.

In the absence of definitive studies proving that aerosols in clinical settings pose no risk of HIV transmission to personnel, surgeons, dentists, and others should avoid techniques which generate such aerosols or even smoke plumes and patients known to be infected with HIV. Where such procedures are unavoidable, personnel should consider using high efficiency filtration masks and systems for containing aerosols.

[References Omitted]

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December 22, 1995/Vol. 44/No. 50

MORBIDITY AND MORTALITY WEEKLY REPORT

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929 Case-Control Study of HIV Seroconversion in Health-Care Workers After Percutaneous Exposure to HIV-Infected Blood--France, United Kingdom and United States, January 1988-August 1994

933 Hypothermia-Related Deaths--New Mexico, October 1993-March 1994

Printed and distributed by the Massachusetts Medical Society, publishers of *The New England Journal of Medicine*

Notice to Readers

Because of the furlough of U.S. government employees, CDC has restricted its activities to responses to emergencies and other public health matters of extreme urgency. This issue of MMWR contains two reports with immediate public health implications: one report provides measures of the prevention of hypothermia-related morbidity and death, and the other report summarizes the findings of a new study of factors associated with risk for and prevention of human immunodeficiency virus (HIV) infection in health-care workers following percutaneous exposure to HIV-infected blood. Other reports of public health importance and findings from the ongoing National Notifiable Disease Surveillance System will be published at a later date, and printed versions of this issue will be available to CDC's subscribers at a later date. The next scheduled issue will be Volume 44, Numbers 51 and 52, dated January 5, 1996, and will include the figure and tables on notifiable diseases for the weeks ending December 16, 23, and 30, 1995.

David Satcher, M.D., Ph.D.
Director, CDC

Case-Control Study of HIV Seroconversion in Health-Care Workers After Percutaneous Exposure to HIV-Infected Blood -- France, United Kingdom, and United States, January 1988 -- August 1994

Health-care workers (HCWs) are potentially at risk for human immunodeficiency virus (HIV) infection through occupational exposures to blood. Although prospective studies

indicate that the estimated risk for HIV infection after a percutaneous exposure to HIV-infected blood is approximately 0.3% (1,2), factors that influence this risk have not been determined. To assess potential risk factors, CDC, in collaboration with French and British public health authorities, conducted a retrospective case-control study using data reported to national surveillance systems in the United States, France, and the United Kingdom. This report describes the study and summarizes results that suggest that risk factors for HIV transmission include certain characteristics

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HIV Seroconversion -- Continued

of the exposure and the source patient; in addition, postexposure use of zidovudine (ZDV) by HCWs was associated with a lower risk for HIV transmission.

Case-HCWs had a documented occupational percutaneous exposure to HIV-infected blood (i.e., a needlestick or a cut with a sharp object [e.g., scalpel or lancet]), HIV seroconversion temporarily associated with the exposure, and no other concurrent exposure to HIV. Control-HCWs had a documented occupational percutaneous exposure to HIV-infected blood and were HIV seronegative at the time of exposure and at least 6 months later. Case-HCWs were identified through reports to national surveillance systems for occupationally acquired HIV infection operated by CDC, in cooperation with state and local health departments (United States), the National Public Health Network (Réseau Naitonal de Santé Publique) (France), and the Public Health Laboratory Service Communicable Disease Surveillance Center (United Kingdom). Control-HCWs were identified through reports to a passive surveillance project maintained by CDC since 1983 that includes data from approximately 300 health-care institutions in the United States(1).

The study included all case-HCWs reported in the United States whose exposure occurred during January 1988-August 1994 and all control-HCWs exposed after January 1988 whose 6-month

follow-up evaluation was completed as of August 1994. Case- and control-HCWs reported in the United States before 1988 were excluded from the analysis because information on some variables was not routinely collected and because postexposure use of ZDV was infrequent before 1988 (1). For similar reasons, analysis was limited to case-HCWs reported in France since 1990 and in the United Kingdom since 1989.

Information obtained about HCWs included age; sex; occupation; work location; and whether postexposure antiretroviral agents were offered, whether they were used, how long after the exposure the first dose was used, daily dosage, and duration of treatment. Information about source patients included stage of HIV infection (acquired immunodeficiency syndrome [AIDS], symptomatic, or asymptomatic), use of antiretroviral drugs at the time of the HCW's exposure, and presence of terminal illness (i.e., death because of AIDS within 2 months after the exposure). Information about exposures included the type of device involved, gauge of hollow-bore needle, type of procedure being performed, whether the procedure was an emergency, use of gloves, time from use of the device to exposure, presence of visible blood from the source patient on the device, and severity of injury. Severity of injury was defined as superficial (surface scratch, no blood appeared), moderate (penetrated skin and blood appeared), or deep (deep puncture or wound with or without bleeding).

The study included 31 case-HCWs (23 from the United States, five from France, and three from the United Kingdom) and 679 control-HCWs (who were from 190 of the reporting health-care institutions). Of the 31 exposures sustained by case-HCWs, 29 (94%) were needlesticks (all with hollow needles) and two (7%) involved other sharp objects. Of the 679 exposures sustained by control-HCWs, 620 (91%) were needlesticks (including 594 hollow and 26 solid needles) and 59 (9%) involved other sharp objects.

For both case- and control-HCWs, 74% were exposed during 1990-1994, when ZDV postexposure use had become more common (1). During 1990-1994, 17 (81%) of

21 case HCWs had been offered ZDV, and from September 1990 (when collection of information on whether ZDV was offered to

control-HCWs became routine) through 1994, 268 (79%) of 338 control-HCWs were offered ZDV. ZDV postexposure prophylaxis was used by nine (29%) case-HCWs and 247 (36%) control-HCWs (crude odds ratio=0.7;95% confidence interval [CI]=0.3-1.7). Regimens for case- and control-HCWs generally were 1000 mg/day for 3-4 weeks; the small number of case-HCWs who used ZDV precluded assessment of differences in ZDV regimens between case- and control-HCWs.

All variables that were statistically significant in the univariate analysis and variables potentially important for prevention (e.g., use of gloves, whether ZDV was offered, and whether ZDV was used) were examined using logistic regression analysis. Based on this analysis, factors associated with HIV transmission included a deep injury, device visibly contaminated with the source patient's blood, procedures involving a needle placed directly in a vein or artery, and terminal illness in the source patient. In addition, case-HCWs were significantly less likely to use ZDV than control-HCWs (adjusted odds ratio=0.2 $p<0.01$) (Table 1). The crude odds ratio for ZDV use differed from the adjusted odds ratio because ZDV use was more frequent, among both case- and control-HCWs, for exposures characterized by the other factors. All factors in the model also were significant when the analysis was restricted to case-HCWs from the United States.

The degree of susceptibility to ZDV of HIV strains from source patients and case-HCWs is unknown. Information about antiretroviral treatment for source patients was available for seven case-HCWs and 124 control-HCWs who had used ZDV; five (71%) case-HCWs and 87 (70%) control-HCWs were exposed to blood from source patients who had been receiving ZDV at the time of the exposure.

Reported by: State and territorial health depts. CDC Cooperative Needlestick Surveillance Group. D Abiteboul, MD, Institute National de Recherche et de Sécuritéand Groupe d' Etude sur

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[1]Information on terminal illness in the source patient was missing for 19% of case-HCWs and 48% of control-HCWs; information on visible blood on device was missing for 3% of case-HCWs and 6% of control-HCWs. By recoding the missing values to zero and including missing value indicator variables for these factors in the model, these HCWs were retained in the analysis and their potential

confounding influence could be assessed. No significant interactions were found among the risk factors in the model or between the risk factors and the missing value indicators. When all HCWs with missing values for any of the factors were excluded from the analysis, all of the factors remained significant, with similar adjusted odds ratios but larger confidence intervals.

TABLE 1. Risk factors for HIV infection in health-care workers after percutaneous exposure to HIV-infected blood, based on a case-control study -- France, United Kingdom, and United States, January 1988-August 1994

Rick Factor	Adjusted odds ratio*	(95% CI[†])
Deep injury	16.1	(6.1-44.6)
Visible blood on device	5.2	(1.8-17.7)
Procedure involving needle placed directly in a vein or artery	5.1	(1.9-14.8)
Terminal illness in source patient	6.4	(2.2-18.9)
Postexposure use of zidovudine	9.2	(0.1- 0.6)

*All were significant at $p<0.01$.
[†]Confidence interval.

UNITED STATES DISTRICT COURT
DISTRICT OF MAINE

SIDNEY ABBOTT,	]	
Plaintiff	]	Civil Action No. 94-0273-B
vs.	]	
RANDON BRAGDON, D.M.D.	]	
Defendant	]	

DECLARATION OF LEON R. KELLEHER, D.D.S.

Pursuant to 28 U.S.C. § 1746, I, Leon R. Kelleher, declare that:

1. I sent a letter dated January 9, 1996, to Dr. Randon E. Bragdon, a true and complete copy of which is attached hereto.

2. I hereby incorporate the complete text of that letter as if fully set forth herein.

I hereby declare under penalty of perjury that the foregoing information is true to the best of my knowledge and belief.

Executed on March 27, 1996.

S/Leon R. Kelleher
Leon R. Kelleher, D.D.S.

State of Illinois
County of , ss.

On this 27 day of March, 1996, before me personally appeared the above named Leon R. Kelleher and subscribed to the foregoing.

OFFICIAL SEAL
KATHRYN A. SEIDEL
Notary Public State of Illinois
My Commission Exp. Mar. 9, 1998

S/Kathryn A. Seidel
Notary Public
Print Name: Kathryn A. Seidel

[App. 441]

PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER 1653 West Congress Parkway,
UNIVERSITY Chicago, Illinois 60612-3864 - 312-942-5000

RUSH
SECTION OF DENTISTRY
Leon R. Kelleher, DDS, Director

January 9, 1996

Dr. Randon E. Bragdon
30 Judson Boulevard
Bangor, Maine 04401

Dear Dr. Bragdon.

We read with appropriate concern the recent (1-8-96) article in the ADA News regarding your alleged discrimination in the case of the HIV positive patient whom you proposed to treat in a hospital operatory rather than your office in order to limit the risk of exposure to yourself and your staff. By this time we are certain that you have received letters of support for your position as well as some which are anything but that. Please allow us to add our names to the former list.

Our staff of seventeen dentists practices in a private intramural group which treats more than a hundred known HIV+ patients. We share many of your concerns and frequently utilize "special facilities" or measure to assure our safety as well as that of the patient. Let me begin by assuring you that your obvious concerns as to whether the CDC Guidelines regarding universal precautions are adequate to protect operator as well as patient do have merit. We invite your attention to the enclosed study by Drs Robinson and Johnson of Stanford University which clearly shows that aerosols generated during the use of certain surgical instruments are contaminated. We are currently designing a study which is intended to show that the HIV can and does contaminate the aerosol generated by the dental high speed handpiece and the ultrasonic

cavitron. Until the results of this study are ready for publication we do not intend to rely upon the assurances of the CDC or anyone else who purports that immersing ones head into an infected aerosol is perfectly safe since it cannot be unequivocally proven that anyone has contracted HIV+ in that manner.

In addition to the use of published universal precautions, our standard operating procedure is to avoid the use of aerosol producing instrumentation in known HIV+ cases. Patients who require dental prophylaxis are hand-scaled rather than cleaned ultrasonically. Patients on whom the use of the high speed handpiece is unavoidable are done with high volume evacuation under laminar flow conditions with hood or full face protection and remote air source for the operator. These procedures, of course, do not protect against the HIV+ patient who denies that status on his

[App. 442]

medical history or who does not yet know of his seroconversion or the patient who is HIV infected and who has not yet seroconverted. The question of whether all patients will eventually require treatment under these conditions remains academic until the results of these studies are finally published, repeated by others, and then disseminated to the profession. There will be no measure of consolation in being the first dentist to contract AIDS through a contaminated aerosol.

It is somehow ironic that the very patients for whom we assume the most risk are usually the ones who respond to that humanitarian gesture with a lawsuit. One can only wonder whether those special interest groups whose primary banner is political correctness and whose efforts are usually behind the filing of such litigation will be there to support the cause of the first dentist or hygienist who contracts AIDS from treatment of a patient and is denied disability coverage by his or her insurer!

Good luck with your appeal.

Respectfully,

S/Leon R. Kelleher, DDS.

Dr. Leon R. Kelleher
Director
Section of Dentistry

LrK/sar
encl. (1)

[App. 443]

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Plaintiffs-Appellees

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant

ON REMAND FROM THE UNITED STATES SUPREME COURT

RESPONSE TO ABBOTT'S BRIEF

On pages 16-17 of her brief, Abbott presents her argument that the language used in CDC publications supports her contention that CDC recommended universal precautions are so effective that they are the maximum desirable or reasonable precautions for dentists performing invasive procedures on persons infected with HIV. Her principal argument relies on a quotation from CDC's 1987 *Recommendations for Prevention of HIV Transmission in Health-Care Settings*, MMWR, vol. 36, no. 2S (1987). Portions of that CDC publications are reproduced on Addendum pages 160-162 of Dr. Bragdon's blue brief, and are attached to this response as Exhibit A. Highlighted on Exhibit A is the portion of this publication quoted by Abbott on page 17 of her brief. The language quoted by Abbott does not pertain to invasive procedures, which are defined in the next paragraph. Highlighted on Exhibit A in the next paragraph is the CDC's statement: "The universal blood and body-fluid precautions listed above, combined with the precautions listed below, ***should be the minimum precautions*** for all such invasive procedures" (emphasis added). Invasive procedures are defined in that paragraph to include "the manipulation, cutting, or removal of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists." Filling a cavity near the gum line, such as Abbott's, is a perfect example of

an invasive procedure. *E.G.* App. 192-195. This alone refutes Abbott's thesis, and is another illustration of how Abbott's arguments are politics masquerading as science. Summary judgment should therefore be awarded to Dr. Bragdon, since based on this CDC publication the only reasonable conclusion is that universal precautions ***"should be the minimum precautions"*** (emphasis added). In the alternative, Dr. Bragdon clearly raises a genuine issue for trial.

Date: October 5, 1998

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[Excerpts from CDC, *Recommendations for Prevention of HIV Transmission in Health-Care Settings*, 36 MMWR No. 2S (August 21, 1987)]

Exhibit A to Response to Abbott's Brief

5. Health-care workers who have exudative lesions or weeping dermatitis should refrain from all direct patient care and from handling patient-care equipment until the condition resolves.

6. Pregnant health-care workers are not known to be at greater risk of contracting HIV infection than health-care workers who are not pregnant; however, if a health-care worker develops HIV infection during pregnancy, the infant is at risk of infection resulting from perinatal transmission. Because of this risk, pregnant health-care workers should be especially familiar with and strictly adhere to precautions to minimize the risk of HIV transmission.

Implementation of universal blood and body-fluid precautions for **all** patients eliminates the need for use of the isolation category of "Blood and Body Fluid Precautions" previously recommended by CDC (7) for patients known or suspected to be infected with blood-borne pathogens. Isolation precautions (e.g., enteric, "AFB" [7]) should be used as necessary if associated conditions, such as infectious diarrhea or tuberculosis, are diagnosed or suspected.

Precautions for Invasive Procedures

In this document, an invasive procedure is defined as surgical entry into tissues, cavities, or organs or repair of major traumatic injuries 1) in an operating or delivery room, emergency department, or outpatient setting, including both physicians' and dentists' offices; 2) cardiac catheterization and angiographic procedures; 3) a vaginal or cesarean delivery or other invasive obstetric procedure during which bleeding may occur, or 4) the manipulation, cutting, or removal of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists. The universal blood and body-fluid precautions listed above,

combined with the precautions listed below, should be the minimum precautions for **all** such invasive procedures.

1. All health-care workers who participate in invasive procedures must routinely use appropriate barrier precautions to prevent skin and mucous-membrane contact with blood and other body fluids of all patients. Gloves and surgical masks must be worn for all invasive procedures. Protective eyewear or face shields should be worn for procedures that commonly result in the generation of droplets, splashing of blood or other body fluids, or the generation of bone chips. Gowns or aprons made of materials that provide an effective barrier should be worn during invasive procedures that are likely to result in the splashing of blood or other body fluids. All health-care workers who perform or assist in vaginal or cesarean deliveries should wear gloves and gowns when handling the placenta or the infant until blood and amniotic fluid have been removed from the infant's skin and should wear gloves during post-delivery care of the umbilical cord.

2. If a glove is torn or a needlestick or other injury occurs, the glove should be removed and a new glove used as promptly as patient safety permits; the needle or instrument involved in the incident should also be removed from the sterile field.

Precautions for Dentistry

Blood, saliva, and gingival fluid from **all** dental patients should be considered infective. Special emphasis should be placed on the following precautions for preventing transmission of blood-borne pathogens in dental practice in both institutional and non-institutional settings.

1. In addition to wearing gloves for contact with oral mucous membranes of all patients, all dental workers should wear surgical masks and protective eyewear or chin-length plastic face shields during dental procedures in which splashing or spattering of blood, saliva, or gingival fluids is likely. Rubber dams, high-speed evacuation, and proper patient positioning,

when appropriate, should be utilized to minimize generation of droplets and spatter.

2. Handpieces should be sterilized after use with each patient, since blood, saliva, or gingival fluid of patients may be aspirated into the handpiece or waterline. Handpieces that cannot be sterilized should at least be flushed, the outside surface cleaned and wiped with a suitable chemical germicide, and then rinsed. Handpieces should be flushed at the beginning of the day and after use with each patient. Manufacturers' recommendations should be followed for use and maintenance of waterlines and check valves and for flushing of handpieces. The same precautions should be used for ultrasonic scalers and air/water syringes.

3. Blood and saliva should be thoroughly and carefully cleaned from material that has been used in the mouth (e.g., impression materials, bite registration), especially before polishing and grinding intra-oral devices. Contaminated materials, impressions, and intra-oral devices should also be cleaned and disinfected before being handled in the dental laboratory and before they are placed

---

*General infection-control precautions are more specifically addressed in previous recommendations for infection-control practices for dentistry (8).

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Plaintiffs-Appellees

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant

ON REMAND FROM THE UNITED STATES SUPREME COURT

RENEWED MOTION (1) FOR REMAND FOR FURTHER DISCOVERY AND DEVELOPMENT OF THE RECORD CONCERNING CLAIMS MADE BY THE AMERICAN DENTAL ASSOCIATION AND (2) IN THE ALTERNATIVE, MOTION FOR LEAVE TO RESPOND POINT-BY-POINT TO SUCH CLAIMS TO THE EXTENT FEASIBLE WITHOUT DISCOVERY

In its amicus brief, the American Dental Association (the "Association") makes many self-serving claims concerning (i) the historical origins of its policies on HIV and (ii) what is or is not a valid "scientific" inference and/or the level of risk to dentists. Examples of the former sorts of claims are:

> All that is said above about the Science Council was applicable to these predecessor councils [which formulated the 1987-1991 policies in question] prior to the time of their consolidation [in 1994]." (p.3 n.4)

[T]he Science Council also utilizes professional American Dental Association staff housed in the Division of Science, which presently has 28 highly qualified experts. . . . [T]he Science Council retains numerous consultants and advisors to facilitate its work. Those consultants have historically included experts . . . such as Drs. Donald Marianos, John Molinari, . . . and Deborah Greenspan. (p. 4) Similar resources were in place when the Association's policies on HIV/AIDS were initially developed in 1987. (p. 4)

[T]he Science Council is careful to ensure that studies used in determining policy issues have gone through the scientific review process. (p. 4)

The development of the Association's policies regarding HIV-disease, as with other policies based on the recommendations of the Science Council and its predecessors, has been scientifically driven. (p. 5)

Based on this [i.e. the 10 "bulleted" items listed on pages 10-11] and other scientific evidence [not specified], the Association [the entire organization?] believed [in 1987] the risk of occupationally acquiring HIV-infection through the practice of dentistry to be so low as to be almost undetectable. (p. 11)

[The Association (or some committee?) actually reviewed the CDC reports of 7 possible transmissions to dentists, but decided that] these "possible" cases [did not provide] a scientific basis to change its policies, especially given the inaccuracies often associated with sexual histories. (p. 12 n.27)

> While the Association's policy and ethics code are not coterminous, each is driven by science. (p. 18)

Examples of the latter are:

> The anti-HIV-1 activity of saliva probably explains the virtual absence of oral transmissions of HIV. (p. 14)
>
> [Results from the Association's "Health Screening Program"] attest to the safety of treating patients with HIV-disease, and that the risk of occupationally acquiring HIV-infection through the practice of dentistry is so low as to be almost undetectable. (p. 14)

Dr. Bragdon has no way of responding in a meaningful way to most of these sorts of claims unless the Court orders a remand. For example, the Association claims that "[a]ll that is said above [i.e., in the text of the Association's brief above footnote four] about the Science Council was applicable to these predecessor councils [which formulated the 1987-1991 policies in question]." (p. 3 n.4) Very little, actually, is said about the Science Council in the Association's brief above footnote four, whereas a great deal is said below footnote four about the alleged scientific methods of the Science Council. Is this just a typographical error, so that what is said below the note also applies to the predecessor councils? Or is this a lawyer's way of saying something that is true (namely, only what is said above is accurate for pre-1994 councils), while nonetheless giving the impression, unless one reads carefully, that there is no significant difference between the Science Council and predecessor councils? In addition, as far as Dr. Bragdon can see, the Association does not even specify which predecessor council or councils was responsible for researching the matter and making recommendations to the Board of Trustees (the "Board"). Rather than shedding any light on the actual process, the Association vaguely seems to imply that the original recommendations to the Board, where ever they came from, were expressly based on the 10 numbered bullets on pages 10-11. But how do we know that it was

not lawyers in 1998 who put these bullets together? Every one of the 10 bullets is simply a paraphrase or some spin on some snippet from a CDC publication between 1983 and 1987. All of these publications would certainly have been considered by any committee if it had done anything at all. What, then, does it really mean when the Association says in 1998 that "[b]ased on this and other scientific evidence, the Association [the Board? some committee? a majority of members?] believed the risk of occupationally acquiring HIV-infection through the practice of dentistry to be so low as to be almost undetectable"? And why shouldn't the CDC publications speak for themselves on this subject, since we still have no idea what "expert" is offering his, her, or their spin on the CDC documents? If the Association is correct that some of the experts consulted by the Association are the same experts hired by Ms. Abbott and the United States in this case (e.g., Drs. Greenspan and Molinari), how do we know that this supposed independent analysis is anything but the disguised opinions of Drs. Greenspan and Molinari? Furthermore, was the conclusion that the risk is "so low as to be almost undetectable" ever published or subject to peer review? Is that what the committee told the Board? Then why wasn't that included in the policy statement?

And what of the Board's involvement? Precious little is said by the Association about the Board's involvement in all of this, except to say that "Policy recommendations proposed by Councils are subject to Board of Trustees review and recommendation, which are then passed along to the House for deliberation." (p. 3) It is common knowledge that if a process is not already tainted with politics, sexual or otherwise, the prejudices and inclinations of a governing board or high ranking staff can easily determine the outcome. Dr. Bragdon has reason to believe that there were extreme political biases at work because of the unique composition and experiences of key Association staff members, but without discovery, this can neither be proved nor disproved.

And what about the Health Screening Program? How reliable is its methods? What are its methods? When the Association says that a total of 16,500 dentists have been tested, does that mean 16,500 different dentists have been tested, or that

some core group of roughly 1,000 dentists keep coming back to the annual convention each year and have been tested numerous times? Attachment 2 suggests the latter, since it identifies one person as the "same person" tested in both 1989 and 1990, but we cannot tell. Is this a random sampling and has it been subjected to any rigorous statistical analysis? One would think if it had, the Association would tell us.

Moreover, the Association refers on page 12 to a study "published" in 1992 by its Health Foundation scientists. Is this the same study the Association tried to sell to OSHA in an effort to convince OSHA there was no significant risk to dentists? OSHA said in December of 1991 that the Association's "contention that the HIV risk in dentists is lower than that of the general population is confusing and is not supported by the data the [Association] cited in their posthearing comments." 56 Fed. Reg. 64004, 64092 (Dec. 6, 1991). Now the Association seems to be trying to sell the same thing to the Court. And if this study was published, where was it published? In what? The New England Journal of Medicine? JADA?

Furthermore, according to the Association, its policy is based largely on CDC's 1987 *Recommendations for Prevention of HIV Transmission in Health-Care Settings*, MMWR, vol. 36, no. 2S (1987). Portions of that CDC publication are reproduced on Addendum pages 160-162 of Dr. Bragdon's blue brief, and are attached to this motion as Exhibit A. Although the Association and Abbott assert that the import of this CDC publication is that CDC recommended universal precautions are the maximum desirable or reasonable precautions for dentists, they do not alert the Court to the fact that the CDC publication itself says: "The universal blood and body-fluid precautions listed above, combined with the precautions listed below, ***should be the minimum precautions*** for all such invasive procedures" (emphasis added). (Add. 162 and highlighted on last page of Exhibit A) Invasive procedures are defined to include "the manipulation, cutting, or removal of any oral or perioral tissues, including tooth structure, during which bleeding occurs or the potential for bleeding exists." (*Id.*) The Association and Abbott therefore owe the Court an explanation as to how, in their minds, the only reasonable conclusion is that CDC

recommended universal precautions are the *maximum* precautions desirable when performing invasive procedures on persons known to be infected with HIV, when the CDC says they ***"should be the minimum precautions"*** (emphasis added). The Association also owes the Court an explanation as to how, in its mind, there was so much "scientific support" for its views concerning the efficacy of universal precautions, when at the same time two of its high ranking officials, Dr. Neidle and Dr. Burrell, admitted there was no scientific support for the efficacy of universal precautions. (Sept. 28, 1998 Blue Br. Add. 137-141 reproduced at attached Exhibit B)

Dr. Bragdon tried to get answers to these types of questions [e.g., copies of reports from committees to the Board, etc.] from the Association when he learned it was filing a brief in this case. (Sept. 28, 1998 Blue Br. Add. 185-86 reproduced at attached Exhibit C) The Association did not respond to Dr. Bragdon's request, even by way of saying that it would not respond. (Add. 187 reproduced at attached Exhibit C) Finally, when Dr. Bragdon conditioned consent to the Association's amicus brief on at least a response indicating either the Association would honor the request or for whatever reason refuse to do so, the Association refused to provide information of the sort one would think it could easily produce (assuming there was any sophisticated scientific research, reports, analyses, or the like) and would have used in writing its brief. (Add. 190 reproduced at attached Exhibit C) That is why Dr. Bragdon cannot respond in a meaningful way to most of the claims made by the Association concerning the historical development of its policies, or answer many of the questions raised above, unless the Court orders a remand.

In the alternative, Dr. Bragdon at least deserves the right to respond point-by-point to those arguments made by the Association in its brief which are susceptible to refutation without additional discovery. This would include a point-by-point critique of the inferences (implied and otherwise) drawn by the Association based on the 10 bullets listed on pages 10 to 11, as well as a review of what the numerous publications referred to by the Association in Part V of its brief actually say. The Association implies that some or many of these sources state there is no direct threat, or explain why there is no direct threat. Dr. Bragdon deserves the right to

demonstrate otherwise. In addition, because the Association cannot be expected to cooperate voluntarily, Dr. Bragdon should be allowed at least several months to assemble through his own resources what the Association refers to as "[a]ll of these materials" (p. 19), or at least as much as practical in that time, in order to refute what Dr. Bragdon believes are the Association's unsupported and exaggerated claims.

WHEREFORE, Dr. Bragdon renews his motion for a remand for further discovery and development of the record on the import and admissibility of the Association's policies and their purported scientific basis. In the alternative, Dr. Bragdon requests leave to file a response to the Association's brief not to exceed forty pages to be filed on or before 60 days from the issuance of an order permitting the response.

Date: October 7, 1998

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John W. McCarthy

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[EXHIBITS A, B and C omitted]

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[P. 297] (Whereupon, at 3:52 p.m. a recess until 4:10 p.m.)

VICE CHAIRPERSON REYNOSO: We are ready to proceed. This is our last panel this afternoon. We're very appreciative of your being here. In this panel we will be discussing the AIDS/HIV issues and the ADA in light of the recent Supreme Court case law. Among the topics that may be addressed are the Centers for Disease Control guidelines on occupational transmission of HIV, evaluating the "direct threat" defense, and work place policies of AIDS/HIV infected employees. So it will be a very timely topic for us to hear about and I think important in terms of the evolution of the ADA. With that, Madam General Counsel, would you call the panel.

GENERAL COUNSEL MOORE: Thank you. Would you all stand to be sworn.

VICE CHAIRPERSON REYNOSO: Would you please raise your right hand. Do you swear to affirm that you will testify truthfully to the best of your abilities? If you do, please respond by saying I do.

ALL: I do.

****

[P. 299] DR. VALDISERRI: I'm Ron Valdiserri. I'm the Deputy Director of the National Center for HIV, STD, and TB prevention at the Center for Disease Control and Prevention in Atlanta, Georgia.

****

[P. 339] DR. VALDISERRI: If I might comment, there are a number of issues brought up related to that in terms of surveillance. First of all, let me remind the commission that the

condition of AIDS is reported by every state and territorial health department in the United States to CDC. That's different from the situation of HIV reporting. There are, I believe, 32 at present states who report HIV infection whether or not the individual has clinical AIDS or not.

From one point of view I agree with my colleague that AIDS surveillance gives us more information about the end stage, if you will, of infection rather than some of the earlier aspects of infection. However, let me add that the AIDS surveillance system is widely regarded by epidemiology experts all around the world. They often refer to it as the Cadillac of surveillance with extremely high rates of completeness of reporting and accurate information. I do not believe, nor does CDC believe, that we are missing large amounts of information. What our concern has been actually has been trying to get information about earlier stages of the epidemic.

I do want to just clarify when the figures were given about the cases of occupational transmission. Again, we're talking about transmission from patients to health care worker and I said that a certain number were definite and then 132 or so were probable. What that means is that after extensive investigation by CDC the health care workers were found to -- you know, there was a documented occupational episode that could have transmitted virus from an infected patient. The health care workers were not found to have another risk behavior that would expose them to the virus but we did not have a negative tested base line. All we know is that at some point off in the future they were infected. That's why they are referred to as probable.

****

UNITED STATES COURT OF APPEALS
FOR THE FIRST CIRCUIT

No. 96-1643

SIDNEY ABBOTT, ET AL.,
Plaintiffs-Appellees

v.

RANDON BRAGDON, D.M.D.,
Defendant-Appellant

BEFORE
SELYEA, Circuit Judge,
ALDRICH, Senior Circuit Judge,
AND STAHL, Circuit Judge.

ORDER OF COURT

Entered: October 21, 1998

It is ordered that appellant's renewed motion to remand be denied, without prejudice, to its renewal at the time of oral argument;

Leave is granted the American Dental Association's to file an amicus brief;

Appellant's motion to file a two-page response is granted; and

It is further ordered that we treat Bragdon's motion as his response to the American Dental Association's amicus brief and hereby deny his alternative motion for further briefing.

This case is scheduled for oral argument on Wednesday, December 9, 1998.

By the Court:

Phoebe D. Morse, Clerk

By: Janice M. O'Neil
Chief Deputy Clerk

No. 98-1536

Supreme Court, U.S.
FILED
APR 23 1999
CLERK

In The

Supreme Court of the United States

October Term, 1998

RANDON BRAGDON, D.M.D.,

Petitioner,

v.

SIDNEY ABBOTT, et al.,

Respondents.

On Petition For A Writ Of Certiorari To The United States Court Of Appeals For The First Circuit

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STATUTES CITED:

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STATEMENT OF THE CASE

This action under the Americans with Disabilities Act (ADA) involves petitioner Randon Bragdon, D.M.D.'s refusal to provide routine dental care on September 16, 1994 to respondent Sidney Abbott, solely because she is infected with the Human Immunodeficiency Virus (HIV). The Complaint, filed on December 1, 1994, alleged that Bragdon violated Title III of the ADA, which prohibits public accommodations from discriminating on the basis of disability. Petitioner has claimed throughout this case that he was entitled to refuse treatment to Ms. Abbott in his office because doing so would pose a "direct threat to the health or safety of others." 42 U.S.C. Sec. 12182.

The United States and the Maine Human Rights Commission intervened in support of Ms. Abbott. After discovery was concluded in the district court, all parties simultaneously filed cross motions for summary judgment on all liability issues. Contrary to Bragdon's current assertion to this Court that the issue of "direct threat" must be decided at trial (Petition at 24-25), he argued to the district court that he was entitled to summary judgment on that issue as a matter of law. *See* Motion by Randon Bragdon for Summary Judgment, September 19, 1995.[1]

---

[1] In addition, at oral argument on the summary judgment motions, Bragdon's counsel represented to the district court that it was defendant's position "[a]bsolutely" that the case "can be resolved as a matter of law by summary judgment *one way or the other*" and "[t]hat there is no factual issue to be resolved." *See* Transcript of October 26, 1995 hearing in the district court, Court of Appeals No. 96-1643, Appendix at 439 (emphasis added).

On December 22, 1995, the district court denied Bragdon's summary judgment motion and granted summary judgment in favor of Ms. Abbott and the plaintiff-intervenors. *See Abbott v. Bragdon*, 912 F.Supp. 580, 588-589 (D. Me. 1995) (concluding, among other things, that a thorough review of the record demonstrated that Bragdon had presented only "speculation," "conjecture," and "tangential statistics" to support assertion of direct threat). The Court of Appeals affirmed. *See Abbott v. Bragdon*, 107 F.3d 934, 948 (1st Cir. 1997) (similarly finding that "[e]ach piece of [Bragdon's] evidence is too speculative or too tangential (or, in some instances, both) to create a genuine issue of material fact."). Bragdon never claimed that a trial was necessary on "direct threat" until after the district court had ruled against him on the cross-motions for summary judgment.

Last term, this Court granted certiorari on whether plaintiff had a disability and, if so, whether courts should defer to a health care provider's own judgment when determining whether the provision of care is a "direct threat to the health or safety of others" under Title III of the ADA. *See Bragdon v. Abbott*, ___ U.S. ___, 118 S.Ct. 2196, 2209-2210 (1998). This Court concluded that Ms. Abbott had a disability and that no deference should be granted to the defendant-health care worker in determining the existence of a direct threat. *See* 118 S.Ct. at 2207, 2210-2211.[2]

---

[2] This Court concluded that the Court of Appeals correctly refused to give weight to petitioner's offer to treat Ms. Abbott in a hospital. 118 S.Ct. at 2211. Undisputed testimony in the district court demonstrated that Bragdon never had hospital

In discussing the direct threat issue, this Court explicitly stated that a health care worker, such as Bragdon, has a "duty to assess the risk of infection based on the objective, scientific information." *Id.* at 2210. Although the Court had declined to grant certiorari on whether petitioner raised a "genuine issue of fact for trial" as to the existence of a direct threat, the Court nevertheless "doubt[ed] whether petitioner advanced sufficient evidence to raise a triable issue of fact on the significance of the risk." *Id.* at 2210, 2212. Questioning whether the Court of Appeals gave the correct weight to some pieces of evidence in the record, this Court remanded the case solely to:

> . . . give the Court of Appeals the opportunity to determine whether our analysis of some of the studies cited by the parties would change its conclusion that petitioner presented neither objective evidence nor a triable issue of fact on the question of risk. In remanding the case, we do not foreclose the possibility that the Court of Appeals may reach the same conclusion it did earlier.

Id. at 2213.[3] This Court noted that the purpose of the remand was to "place a complex factual record in proper

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privileges, and that it is equally safe to treat a dental patient with HIV in a private dental office as in a hospital setting. *See id.*; Ms. Abbott's Brief in No. 97-156 at p. 3 n.5; and Ms. Abbott's Brief in Opposition to Petition for Writ of Certiorari in No. 97-156, p. 3 and n.3.

[3] A majority of this Court held that there was no need for a trial on the direct threat issue. *See* 118 S.Ct. at 2213 (Concurring opinion by Justices Stevens and Breyer finding that petitioner had not sustained his burden to raise a triable issue on direct threat, with Justices Kennedy, Souter and Ginsburg concluding

perspective," not to raise or reconsider any question of law. *Id.* at 2213.

On August 14, 1998, the Court of Appeals ordered that the parties simultaneously file supplemental briefs. Subsequently, the First Circuit granted Bragdon's motion to exceed the page limits set by the Court and to file a reply brief. The Court of Appeals heard oral argument on December 9, 1998.

In reviewing the entire record in light of the evidentiary questions posed by the remand, the Court of Appeals found that Ms. Abbott had met her burden of production as the moving party on summary judgment on multiple independent grounds. *Abbott v. Bragdon*, 163 F.3d 87, 89 (1st Cir. 1998). The Court further found that Bragdon's evidence was "still 'too speculative or too tangential (or, in some instances, both) to create a genuine issue of material fact.' " *Id.* at 90 (*quoting Abbott*, 107 F.3d at 948). Based on this review, the Court reaffirmed summary judgment for Ms. Abbott. *See* 163 F.3d at 89-90.

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that the Court of Appeals "may reach the same conclusion it did earlier.").

REASONS FOR DENYING THE PETITION

PETITIONER ALLEGES ONLY AN ERRONEOUS ASSESSMENT OF THE RECORD ON SUMMARY JUDGMENT.

I. There is No Basis for This Court to Utilize Its Supervisory Powers and Conduct a Factual Review of the Summary Judgment Record.

In his Petition for Certiorari, Bragdon asks this Court to exercise its supervisory powers and review the record supporting the grant of summary judgment. There is no reason or need for this Court to take this extraordinary step and relitigate record-based determinations which have now been made by the lower courts on three occasions. *See* United States Supreme Court Rule 10.

First, the selective pieces of evidence cited in Bragdon's Petition do not establish any error by the Court of Appeals. An examination of the entire summary judgment record is necessary to demonstrate that Bragdon did not present sufficient competent evidence. For example, Bragdon claims that the First Circuit ignored his expert's calculation of the risk. Petition at 7-9. Yet, the full record shows that those calculations made by Bragdon's expert were based on assumptions about dentistry which had no support in the record and thus were not competent evidence. *See, e.g.*, Brief of Plaintiff-Appellee Sidney Abbott in Court of Appeals, dated September 6, 1996, at 42-43. Bragdon's Petition is replete with other incomplete characterizations of the record.[4] The lower courts have now

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[4] In the Petition, Bragdon repeatedly claims that there is no evidence that universal precautions reduce the risk of HIV

thoroughly explored the summary judgment record *in toto* on three occasions and have found that Bragdon did not present sufficient competent evidence.[5]

Second, the fact that Bragdon disagrees with the outcome of the remand is not a basis for this Court to invoke its supervisory powers. Bragdon had a full opportunity to present evidence to the district court prior to that court's review of the cross motions for summary judgment. Moreover, he was permitted to present additional arguments to the Court of Appeals on remand about the state of the evidence below. The Court of Appeals did not disregard those arguments, it simply disagreed.

Contrary to Bragdon's characterization of the Court of Appeals' decision, that Court found that the 1986, 1987 and 1993 Guidelines and the American Dental Association Policy represent "only a fraction of the proof advanced to support Ms. Abbott's motion." 163 F.3d at 89. Following instructions from this Court, the Court of Appeals determined "testimony from numerous health experts [on behalf of Ms. Abbott] indicating that it is safe to treat patients with HIV in dental offices" was based on evidence available in September 1994. *See* 118 S.Ct. at 2212; 163 F.3d at 89. Moreover, the Court of Appeals

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transmission. Petition at 9-11. The Court of Appeals, however, determined that such a statement alone provides no probative evidence about the baseline level of risk. 107 F.3d at 948.

[5] Faced with his failure to meet his burden on summary judgment, Bragdon, without justification, now impugns the motives of the Court of Appeals. *See* Petition at 5 (claiming that the opinion of the Court of Appeals carries a "policy determination" that regardless of the evidence, "the case should not go to trial.").

found that the opinions of several prominent experts on behalf of Ms. Abbott about such evidence available in 1994 "likely suffice to prove Ms. Abbott's point." *See* 163 F.3d at 89. At the same time, the Court re-examined Bragdon's evidence, including the evidence about the CDC's categorization of seven "possible" cases of patient-to-dental worker HIV transmission. 163 F.3d at 89-90. Basing its review on this Court's analysis of the significance of that evidence,[6] the Court of Appeals found that the record established that the CDC's definition of "possible" cases was well known prior to 1994. *Id.* at 90. As a result, in accordance with this Court's instructions, the Court of Appeals found that those cases did not create a genuine dispute of material fact. *Id.*

II. The Petition Inappropriately Cites Post-1994 Evidence as Well as Evidence Not in the Record.

In attempting to establish factual disputes, Bragdon has improperly sought to rely on evidence that was not in the record or which post-dated the discriminatory denial of care. *See* 118 S.Ct. at 2211; 107 F.3d at 944. For example, on remand Bragdon attempted to introduce a statement of a CDC official made in 1998 characterizing the "possible" category of occupational transmissions as "probable." Petition at 15-16. In fact, the transcript of the testimony of Ronald Valdiserri, M.D. before the United

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[6] In discussing this evidence, Bragdon essentially argues that the Court of Appeals should have disregarded this Court's explicit directions and adopt an interpretation of those cases which Bragdon argued unsuccessfully to this Court last term. Petition at 14-15.

States Civil Rights Commission on November 12, 1998 referred on page 313 to the categories of either "possible" or "documented" occupational transmissions. Dr. Valdiserri later made reference to the same statistics but misstated the categories as "definite" and "probable." To correct his misstatement, Dr. Valdisseri submitted an affidavit explaining that he had misspoken and reiterating that the seven "possible" cases do not represent "probable" cases.[7] After briefing on Bragdon's motion to submit the testimony of Dr. Valdiserri, the Court of Appeals disregarded the November 1998 testimony because it post-dated September 1994, the date which this Court reaffirmed as the critical date for determining direct threat, 118 S.Ct. at 2211-2212, and because it was never a part of the summary judgment record. Moreover, the clear evidence was that Dr. Valdiserri simply misspoke, not that he was disagreeing with the CDC's long-standing surveillance definitions.

In his Petition, Bragdon similarly cites numerous other documents which are not in the summary judgment record in this case. *See, e.g.*, 318a, 414a, 418a, and the Journal of the American Dental Association article at page 15 of the Petition. The question before the Court of Appeals, initially and on remand, was whether summary judgment was properly granted on the extensive record established by both parties on cross motions for summary

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[7] *See* United States' Response to Appellant's Motion to Stay Final Decision (with Affidavit of Ronald Valdiserri, M.D.) and Plaintiff-Appellees' Opposition to Appellant's Motion to Stay Final Decision, Court of Appeals, December 7, 1998.

judgment below. Bragdon cannot add to the record now in an attempt to relitigate the issues in this Court.

III. The Court of Appeals' Decision on Remand Raised No Issue of Law.

Although primarily asking this Court to conduct a de novo review of the summary judgment record, Bragdon identifies legal issues which he would like this Court to address. *See* Petition at 17-28. These issues, some of which Bragdon attempted to argue to the Court last term,[8] were not within the scope of the remand and therefore were not reconsidered by the Court of Appeals.

The legal arguments that petitioner now raises have all been heard before in this very case, both by this Court and the Court of Appeals. This Court should not now indulge Bragdon's attempt to relitigate last term's decision and reconsider the meaning of direct threat in the context of this record.

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[8] For example, Bragdon now repeats the argument he made to this Court last year that cases finding that certain HIV-infected health care workers posed a direct threat to patients established that Ms. Abbott posed a direct threat to Bragdon. *See* Petitioner's Reply Brief in 97-156 at 19. From these cases, petitioner claims that there is a split in the circuits justifying the grant of certiorari. However, the applicability of these cases to the instant case was raised before, and there is no justification warranting the reconsideration of the issue.

CONCLUSION

Bragdon has had multiple opportunities to litigate the issues raised in this case. His arguments have received a thorough review, first by the district court, twice by the Court of Appeals, and once by this very Court. There is no basis to reopen this case yet again. For the reasons given above, Respondents Sidney Abbott and the Maine Human Rights Commission respectfully request that this Court deny the Petition for Writ of Certiorari.

Respectfully submitted,

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No. 98-1536

In The

Supreme Court of the United States

October Term, 1998

Randon Bragdon, D.M.D.,

Petitioner,

v.

Sidney Abbott *et al.*,

Respondents,

On Petition for a Writ of Certiorari to the United States Court of Appeals for the First Circuit

Brief of Amicus Curiae of Congressman Charles Norwood, D.D.S., Congressman Tom Coburn, M.D., The American Association of Forensic Dentists, The Association of American Physicians and Surgeons, Dentists for Preservation of Professional Judgment, E.J. Neiburger, D.D.S., Cary Savitch, M.D., Stephen D. Carter, D.D.S., John L. Kennedy D.D.S., Charles E. Smith, D.M.D., Brian E. Eifert, M.D., William J. Dubose, D.M.D, and Robert J. Gherardi, D.M.D in Support of Petitioner Randon Bragdon, D.M.D.

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QUESTIONS PRESENTED

1. Did the First Circuit Court of Appeals err in denying a trial on the merits where there is a question of material fact of whether there is a "direct threat" to the health of the health care worker or other person as determined by the health care professional pursuant to 42 U.S.C. § 12182 (b)(3)?

2. Did the scientific evidence that was available in 1994 evidence a "direct threat" to the health care worker or other person within the meaning of 42 U.S.C. § 12182 (b)(3)?

3. Did the First Circuit Court of Appeals err in denying a trial on the merits which would have produced "current" and "best available" post-1994 evidence of "direct threat" to the health care worker and other person when treating HIV-infected patients pursuant to 42 U.S.C. § 12182 (b)(3)?

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III. Did the First Circuit Court of Appeals err in denying a trial on the merits which would have produced "current" and "best available" post-1994 evidence of "direct threat" to the health care worker and other person when treating HIV-infected patients pursuant to 42 U.S.C. § 12182 (b)(3)? 9

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INTEREST OF THE *AMICI*[1]

This brief represents the interests of Congressman Norwood, who is a dentist, and Congressman Coburn, who is a physician. This brief also represents the interests of dentists, physicians, medical/dental-related professionals and other persons who have a direct professional and health interest in the outcome of this litigation. The outcome is professional to them, because they are health care professionals engaged in rendering health care to their patients, including those infected with HIV. Secondly, and perhaps more importantly, as natural persons, notwithstanding the fact they are health care professionals, they are susceptible themselves to HIV infection when invasively treating patients with HIV infectious disease.

The matters contained in this brief are relevant to this litigation, because the litigation will ultimately be partially responsible for formulating medical standards required of health care professionals when rendering care to HIV-infected persons. Such persons have been found by the court in this case to be disabled under the Americans with Disabilities Act ("ADA") 42 U.S.C. § 12101 (1994). While such persons may be disabled as a matter of law, the guiding principles for application of the law must be pursuant to sound medical and scientific evidence and not the mere conclusions by the First Circuit Court of Appeals in *Abbott* v. *Bragdon*, 163 F.3d 87 (1st Cir., 1998).

The potential infectious condition of patients constitutes a "direct threat" to the persons who make up these *Amici*. It is in the interest of these *Amici* to ask the United States

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[1] The parties have consented to the filing of this brief and consents have been filed with the Court. This brief was authored by A. Eric Johnston, Esquire, with the help of *Amici*. It was not authored by counsel for any party. No person or entity other than *Amici* have made any monetary contribution hereto.

Supreme Court to accept the *Writ of Certiorari* of Dr. Randon Bragdon, which asks for a trial to determine the material facts concerning the "direct threat" issue in this case.

SUMMARY OF ARGUMENT

This brief concerns itself with three aspects of the "direct threat" issue: 1) the legal aspect; 2) the scientific question in 1994; and 3) post-1994 ongoing scientific findings relevant to the issue of "direct threat."

First, there is still a question of material fact concerning the issue of "direct threat." Summary judgment, as exercised by the District Court and confirmed by the First Circuit Court of Appeals, denied a trial where scientific questions of fact can be investigated to the necessary depth. *Abbott* v. *Bragdon*, ibid; *Abbott* v. *Bragdon*, 107 F.3d 934 (1st Cir. 1997).

Secondly, science in 1994 supported the reasonable professional judgment of Dr. Bragdon when he recommended to Ms. Abbott that her invasive dental healthcare treatment be performed in the hospital operating room.

Thirdly, if this case is remanded for trial, not only will substantive, in-depth evidence of science and medicine be adduced, but important post-1994 medical information on "direct threat" not heretofore considered by the Court will be brought to the Court's attention.

Most of the scientific submissions for this brief are extensive and divided into four categories located in the Appendices. The categories represent the health interests of all four groups affected by the issue of "direct threat": 1)

dental patients with HIV infectious disease; 2) other non-infected dental patients; 3) dental assistants/hygienists; and 4) dentists themselves.

By recommending that any patient with HIV infectious disease, regardless of the stage of the disease, have invasive dental healthcare procedures performed in the hospital operating room, this brief argues that Dr. Bragdon was prudent in 1994—and would still be today.

ARGUMENT

Contrary to the Centers for Disease Control and Prevention's ("CDC") claim that "universal precautions" are totally sufficient to prevent the spread of disease, there are significant inadequacies in those precautions. Also, the "passive surveillance" system used by the CDC can only contribute to deficient sampling and incorrect scientific conclusions. Though universal precautions are prudent, they should only constitute the minimal infection control guidelines, instead of the maximum.

I. Did the First Circuit Court of Appeals err in denying a trial on the merits where there is a question of material fact of whether there is a "direct threat" to the health of the health care worker or other person as determined by the health care professional pursuant to 42 U.S.C. § 12182 (b)(3)?

On June 25, 1998, the U.S. Supreme Court remanded the "direct threat" issue to the U.S. First Circuit Court of Appeals to determine, as a matter of fact, whether there is a "direct threat" when a dentist invasively treats a known patient with HIV infectious disease. *Bragdon* v. *Abbott* ___ U.S. ___ 118 S.Ct. 2196, 141 L.Ed. 2d 540 (1998). On December 29, 1998, the First Circuit Court of Appeals ruled

on the case as a matter of law, by affirming previous summary judgment, rather than returning it to the trial court to examine the overwhelming scientific evidence related to the case. *Abbott* v. *Bragdon*, 163 F. 3d 87 (1st Cir. 1998).

The First Circuit Court of Appeals says there is no question of material fact in dispute. There is no evidence in its December 29, 1998, written judgment that any of the scientific findings submitted in three separate briefs filed on behalf of Dr. Bragdon were even considered.

The standards for Summary Judgment are well known. *Celetex* Corp v. *Catrett*, 477 U.S. 317, 106 S. Ct. 2548, 91 L.Ed. 2d. 264 (1986). In this litigation the Courts have addressed the legal issues, but not sufficiently the factual issues. As a matter of law, summary judgment includes a factual finding aspect. That aspect has not been addressed by the First Circuit Court of Appeals. It has ruled only on the written filings from the summary judgment proceeding.

The Circuit Court's ruling on remand, *Abbott* v. *Bragdon*, ibid, merely reviewed the CDC guidelines and American Dental Association policies without taking evidence as to their scientific accuracy and support in the medical community. While the guidelines and policies are themselves evidence, there should be thorough and sifting examination and cross-examination of all evidence in order to determine scientific and medical accuracy. Legal niceties cannot be observed with final and binding judgments based on conclusions when they address a significant and life-threatening disease and protective procedures.

The appendices hereto suggest the significant scientific information available for the Courts to make complete and necessary findings so that medical science and health care are not compromised by legal procedures. The applicable

regulations seek to avoid such mistakes by requiring that a determination of whether there is a direct threat be based on "current medical knowledge or on the best available objective evidence." 28 C.F.R. 36.208 (c) (1996).

The initiating event was Ms. Abbott's 1994 visit to Dr. Bragdon. This is not a damage case for what occurred at that time; rather it was the initiation of a legal determination of, in effect, an applicable standard of care to be observed by health care professionals. Therefore, the time cannot be related to 1994 and earlier, but post-1994 medical evidence must be submitted. Consequently, based on the directions of this Court on its remand, a full and complete trial must be had for the examination of material facts, so the public and health care professionals will be served in the future.

The Court is reminded of Justice Ginsburg's concurring statement:

> I further agree, in view of the "importance [of the issue] to health care workers," *ante*, at 2213, that it is wise to remand, erring, if at all, on the side of caution. By taking this course, the Court insures a fully informed determination whether respondent Abbott's disease posed "a significant risk to the health or safety of [petitioner Bragdon] which [could not] be eliminated by a modification of policies, practices, or procedures...." 42 USC 12182(b)(3). *Bragdon* v. *Abbott*, 118 S.Ct. 2196, 2214 (1998).

II. Did the scientific evidence that was available in 1994 evidence a "direct threat" to the health care worker or other person within the meaning of 42 U.S.C. § 12182 (b)(3)?

The First Circuit Court of Appeals ruled there is not a "direct threat" when a dentist invasively treats a known

patient with HIV infectious disease. However, the scientific evidence proves otherwise.

"Universal precautions" are infection control guidelines recommended by CDC and the American Dental Association. Scientific findings proving the ineffectiveness of latex gloves (recommended as part of universal precautions) demonstrated that there exists a small but significant "degree of risk" when treating patients with HIV infectious disease in the dental office. Latex gloves are porous and can leak.[2] There are numerous cases of Hepatitis B viruses (HBV) passing through latex gloves from health care worker to patient and from patient to health care worker.[3] HBV and HIV are very similar. Latex gloves cannot stop a needle-stick or other sharp dental instruments from injuring the hand. *This is the most common cause of occupational AIDS transmission.*[4] The most recent brief filed by Dr. Bragdon to the First Circuit Court of Appeals demonstrates the significance and prevalence of needle-stick injuries to the dentist.[5] This information was from Dr. David Bell (the CDC's Director of the HIV Infection Programs) and Dr. Sanford Kuvin (Dr. Bragdon's expert witness). The risk of a dentist contracting HIV infectious disease is in the range of 1 in every 67,000 shots (intra-oral injections) administered to

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[2] Lattau, L. et al. "Transmission of Hepatitis B with Resultant Restriction of Surgical Practice." *Journal of the American Medical Association.* 255. February 21, 1986, page 934.

[3] Reingold, Authur, L. et al. "Failure of gloves and other protective devices to prevent the transmission of Hepatitis B Virus to oral surgeons." *Journal of the American Medical Association.* 259. May 6, 1988, pages 2558-2560.

[4] "Occupational Exposure to Bloodborne Pathogens: Final Rule." *Federal Register.* Vol.. 56, No. 235. Dec. 6, 1991, page 64016.

[5] *Amicus* Brief of Defendant/Appellant Randon Bragdon, D.M.D., to the United States Court of Appeals for the First District, on Remand from the United States Supreme Court, pages 5-6.

patients. Those in the medical and dental profession who are aware of these facts believe this is a significant statistic.

Universal precautions were also intended to protect the non-infected dental patient from HIV infectious disease. Dr. Bragdon dealt with the concern for the safety of his other non-infected patients when planning his treatment for Ms. Abbott. Since Ms. Abbott was *a known patient with HIV infectious disease*, Dr. Bragdon concluded that to treat Ms. Abbott utilizing universal precautions in the hospital operating room would not only *reduce* the possibility of patient-to-patient HIV infection, but effectively *eliminate* it.

In 1994, Dr. Bragdon was also faced with a persuasive recommendation—the 1991 OSHA recommendation that "universal precautions" are not sufficient to protect dental health care worker-employees, and prudent additional precautions should be utilized.[6] This OSHA recommendation is still in effect. Dr. Bragdon considered the safety of his dental assistant. He was correct that the distractions and required stressful time schedules of daily private dental practice could be altered in the hospital operating room environment. This option would be safer, because potential needle-sticks and sharp instrument injuries to his dental assistant could be minimized.

Dr. Bragdon also considered his own safety for the same reasons he considered the safety of his dental assistant. He loves his family. Their welfare is at stake when his safety is at stake. It is always prudent to further reduce the degree of risk when possible.

The CDC formulates universal precaution guideline recommendations not by comprehensive and complete

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[6] "Occupational Exposure to Bloodborne Pathogens: Final Rule." *Federal Register.* Vol. 56, No. 235. Dec. 6, 1991, page 64088.

scientific epidemiological studies, but by their admitted irregular and unreliable system of accumulating information, called "passive surveillance." The *ADA News* (American Dental Association) reported that "CDC officials cite the shortcomings of the current AIDS surveillance system.... Minus a clear and full picture of HIV trends, government officials cannot devise effective prevention and treatment programs."[7] The CDC accepts any information that comes its way, but has no comprehensive or scientific method of obtaining data in which to formulate anything more than a *recommendation* of proposed infection control guidelines. Consequently, the "passive surveillance" system used by CDC accumulates deficient, incomplete and unreliable epidemiological data concerning the "degree of risk" of HIV infection for non-infected patients, dental assistants/ hygienists and dentists. No standard "degree of risk" of HIV infection, based on sound and comprehensive scientific findings, was established in 1994. One has yet to be established by the CDC or the American Dental Association.

The scientific community has used and continues to use various words to describe the "degree of risk"—words such as "small" or "very small." Any small degree of risk to a dentist is significant and should be further reduced when possible because he/she is facing the possibility of terrible suffering and certain premature death by contracting HIV infectious disease from an infected patient.

Dr. Bragdon received a recommendation from a reputable dental professional organization concerning the invasive dental healthcare treatment environment and other additional prudent precautions when invasively treating known patients with HIV infectious disease. In 1992, the American Association of Forensic Dentists had researched

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[7] *ADA News.* Volume 29, No. 6. March 16, 1998, page 19.

the issue of hospital versus private office treatment. The Committee recommended hospital dental treatment and other protective actions if the dentist found this to be in his/her patient's and/or his/her best interests.[8]

III. Did the First Circuit Court of Appeals err in denying a trial on the merits which would have produced "current" and "best available" post-1994 evidence of "direct threat" to the health care worker and other person when treating HIV-infected patients pursuant to 42 U.S.C. § 12182 (b)(3)?

In summary judgment on December 22, 1995, Judge Brody stated, "The position taken by Defendant is not an unreasonable one. The court is sensitive to Defendant's concerns about the possibility of the transmission of HIV infectious disease from patient to him, his staff, and other patients, "*particularly in light of how much science has yet to learn about HIV*" (emphasis added).[9] On March 5, 1997, the First Circuit Court of Appeals stated, "We also recognize that cases of this kind are *fact-sensitive*..." (emphasis added). Justice Ginsburg's concurring statement noted above also states the importance of obtaining all relevant facts. All courts inferred that they were open-minded to science as it related to the direct threat issue and question of material fact. The lack of consideration of the relevant post-1994 ongoing science by the First Circuit Court of Appeals proves otherwise and is in direct opposition to the statutory intent requiring "current" and "best available evidence." 28 C.F.R. 36.201 (c) (1996).

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[8] "New AAFD AIDS/HIV Policy Recommendation." *Journal of the American Association of Forensic Dentists.* Vol. 15s, No. 7-9, page 3.

[9] United States District Court, District of Maine, Ruling in *Sidney Abbott* v. *Randon Bragdon, D.M.D.* Dec. 22, 1995, page 26.

For example, according to a CDC 1996 recommendation, post-exposure prophylactic drugs must be administered within the crucial one-hour window after an occupational exposure to HIV infectious disease in order to reduce by 79% the possibility of contracting HIV.[10] Dr. David K. Henderson of the National Institutes of Health, made the following statement in a recent issue of the *Journal of the American Medical Association*:

> As promptly as feasible, the health care worker should report the exposure to her/his institution's occupational medical department...If prophylaxis is elected, the first dose should be administered as promptly as possible. *We strive to administer the first dose within 1 hour of the exposure* (emphasis added).[11]

Today, the recommendation is still that one should be administered these post-exposure prophylactic drugs as quickly as possible after an occupational exposure to HIV infectious disease. Occupational exposures should be considered "urgent" medical concerns to ensure timely administration of post-exposure prophylactic drugs.[12] These drugs are usually readily available in the hospital and are not available in any dental office. Because of the availability of these drugs only in the hospital, it is prudent to invasively treat known patients with HIV infectious disease in the hospital operating room in the event a dentist or dental assistant receives an occupational exposure. Furthermore, the CDC has stated that "The risk per episode of percutaneous

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[10] *Morbidity and Mortality Weekly Report* by the Centers for Disease Control and Prevention. Vol. 45, No. 22. June 7, 1996, page 470.

[11] Henderson, Dr. David K.,"Postexposure Chemoprophylaxis for Occupational Exposures to the Human Immunodeficiency Virus." *Journal of the American Medical Association.* Vol. 281, No.10. 10 March 1999, page 931.

[12] *Morbidity and Mortality Weekly Report* by the Centers for Disease Control and Prevention. Vol. 47, No. RR-7. May 15, 1998, page 1.

injury (e.g., a needle stick) to HIV-infected blood is estimated at 0.4% (upper limit of 95% confidence interval = 0.8%)."[13] This figure could be reduced when performing invasive treatment in a hospital operating room because the advantages of the operating room allow maximum treatment time and concentration.

This post-exposure prophylactic drugs science (post-1994 science) is one of the most important of all discoveries related to the issue of "direct threat", yet it was not considered relevant by the First Circuit Court of Appeals when presented to them in a brief supporting Dr. Bragdon.[14]

CONCLUSION

For the foregoing reasons, it is requested that the U.S. Supreme Court accept Dr. Bragdon's petition for a Writ of *Certiorari* which asks for a trial for the full in-depth investigation of the many scientific findings relevant to the issue of "direct threat."

Respectfully submitted,

A. Eric Johnston, Esq.

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[13] *Morbidity and Mortality Weekly Report* by the Centers for Disease Control and Prevention, Vol. 47, No. RR-17, September 25, 1998, page 3.

[14] *Amicus Curiae* Brief by the American Association of Forensic Dentists to the United States Court of Appeals for the First Circuit. September 29, 1998, pages 9,16,17.

APPENDICES

APPENDIX A

THE INTERESTS OF THE HEALTH OF PATIENTS WITH HIV INFECTIOUS DISEASE WHEN INVASIVE PROCEDURES ARE PERFORMED BY A DENTIST

1) 1994 scientific findings show that Ms. Sidney Abbott was significantly immune-compromised when she visited Dr. Bragdon, proving that Dr. Bragdon was prudent and reasonable in his decision to perform invasive dental procedures on Ms. Abbot in a hospital operating room. These findings were submitted to the First Circuit Court of Appeals in an Amicus *Curiae* Brief in September, 1998.[1]

Dr. Bragdon reported to Judge Morton A. Brody the status of Ms. Abbott's impaired immune system, but this fact was not considered in summary judgment by the United States First Circuit Court of Appeals in its December 29, 1998, ruling. The courts have yet to consider the overall health interests of Ms. Abbott. The courts have only ruled on the civil right, as a matter of law, that gives Ms. Abbott the legal right to be protected from the recommendation of her dentist that she be treated in the cleanest and safest environment in the world—the hospital operating room.

It is acknowledged that the potential for a patient with early onset of HIV infectious disease to acquire an opportunistic infection is small—but it is significant.

The significance of the *consequences* is the issue, not just the "small" probability of a patient with HIV infectious disease

acquiring an opportunistic infection when invasively treated routinely in a dental office as opposed to treatment in the

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[1] Amicus *Curiae* Brief of Dentists for Preservation of Professional Judgement, September, 1998, pages 5-8, 19, 20, 24, 25.

hospital operating room, where infection control is maximized and the potential for opportunistic infections is minimized. The 1994 *Harrison's Principles of Internal Medicine*, thirteenth edition, states:

> It is estimated that approximately 50-70 percent of individuals with HIV infection experience an acute syndrome approximately 3 to 6 weeks following primary infection. Opportunistic infections have been reported during this stage of infection. (Page 1587)
>
> Certain patients will remain entirely asymptomatic despite the fact that their CD+ T cell counts fall to extremely low levels. Certain patients, otherwise asymptomatic, develop persistent generalized lymphadenopathy. With few exceptions, there is a progressive diminution of CD4 + T cell counts during the asymptomatic period, which ultimately leads to a state of immunosupression." (Page 1588)
>
> Neurologic problems occur throughout the course of the disease and may be inflammatory, demyelinating, or degenerative in nature. Virtually all patients with HIV infection have some degree of involvement of the nervous system with HIV. This is evidenced by the fact that cerebrospinal fluid findings are abnormal in approximately 90 percent of patients, even at the asymptomatic stage of HIV infection. (Page 1589)
>
> Candida infections are the most common fungal infections occurring in patients with HIV infection.
>
> Infections are often seen early in the course of HIV infection. (Page 1600)

> Over 95 percent of HIV-infected patients have evidence of infection with hepatitis B: coinfection with hepatitis C and hepatitis D is also quite common. (Page 1607)

> Pulmonary disease is seen in virtually every patient with HIV infection. The most common manifestation of pulmonary disease is pneumonia, and the most common cause of pneumonia is *Pneumocystis carinnii*. In addition to pneumonia, sinusitis is a common respiratory tract complication of HIV infection and is seen at all stages of infection. (Page 1604)

> Hematologic problems are common throughout the course of HIV infection. Anemia is the most common hematologic abnormality seen with HIV infection and is present in 18% of asymptomatic seropositive individuals, 50% of patients with early symptoms, and 75% of patients with AIDS. (Page 1608)

2) Post-1994 Scientific Findings Prove That Ms. Sidney Abbott was significantly immune-compromised when she visited Dr. Bragdon:

In 1994, the risk to Ms. Sidney Abbott's health was well established. We note pages 3 through 6 of the *Amicus Curiae* Brief submitted to the U.S. Supreme Court in *Bragdon* v. *Abbott* by the Infectious Disease Society of America, *et al.* This brief states:

> From the onset of infection, HIV is a serious medical impairment.... Even in patients who are in the early stages of HIV infectious disease, the virus is causing a wide variety of laboratory

> abnormalities.... Moreover, even outwardly asymptomatic patients are at increased risk for a number of very serious medical conditions including mycobacterium tuberculosis, syphilis, acute and chronic hepatitis, toxoplasma *gondii*, and pneumocystis *carininii* pneumonia.

The use of the hospital operating room was a reasonable medical option to protect Ms. Abbott from contracting opportunistic infectious diseases to which she was vulnerable when she visited Dr. Bragdon's office. The American Medical Association's Amicus Brief to the U.S. Supreme Court states:

> This second stage of HIV disease is known as early, or asymptomatic, HIV disease.... A consistent pattern of irregularities in the blood and immune systems can also be detected with laboratory tests. Finally, a range of dermatological abnormalities and oral lesions are not uncommon during this phase. *This stage of the disease requires consistent monitoring and routine medical care to prevent, so far as is practicable, the onset of opportunistic infections* (emphasis added). (pages 7 and 8)*

We have demonstrated our concern that *all* patients with HIV infectious disease be treated with compassion and with current scientific knowledge dictating all treatment plans. This makes the option for invasively treating patients with HIV infectious disease in the hospital operating room reasonable and prudent. This protocol could protect the significantly immune-compromised patient with HIV infectious disease from unnecessary opportunistic infections common in the dental office (e.g. colds, flu, upper respiratory infections, etc).

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* Amicus Curiae Brief of the American Medical Association, at 7, 8, Baragdon b. Abbott, 118 S.Ct. 2196 (1998)

APPENDIX B

THE INTERESTS OF THE HEALTH OF THE NON-INFECTED PATIENTS WHEN A DENTIST TREATS A KNOWN PATIENT WITH HIV INFECTIOUS DISEASE

The CDC and the American Dental Association intend universal precautions to "reduce" the possibility of HIV infection from being contracted by non-infected patients. If a known patient with HIV infectious disease is treated in the hospital operating room instead of the dental office, the possibility of that patient passing HIV infectious disease to a non-infected patient is not reduced, but eliminated. Dr. Bragdon was aware of the relevance of the Acer case. Dr. David Acer gave HIV infectious disease to six of his patients, although he used universal precautions.[2] This case alone caused many dentists to question the effectiveness of universal precautions.

There is also concern about dentists and other dental healthcare workers performing invasive procedures on non-infected patients *without* the non-infected patients having knowledge of the HIV positive status of the dentist.

It is an open policy of the American Dental Association to support the non-disclosure of HIV positive dentists who invasively treat non-infected patients, including children.[3]

The principle of informed consent involves explaining *all* the risks possible when the invasive procedure is performed. We believe the risk, no matter how small, of transferring

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[2] Ciesielski, C. "Transmission of HIV in a Dental Practice." *Annals of Internal Medicine.* Vol. 116, No. 10. May 15, 1992, pages 798-805.
[3] *ADA News.* Vol. 29, No. 4. March 16, 1998, page 19.

HIV infectious disease from a dental health care worker to a non-infected patient should be disclosed before patients give their consent to the invasive procedure. The policy of withholding a dentist's HIV-positive status from the patient is not fair to all concerned. The continual overconfidence and overstatement of the effectiveness of universal precautions has led the American Dental Association to the inappropriate conclusion that dental patients do not have the right to know that their dentists have HIV infectious disease.

Two court rulings have addressed this issue and ruled in favor of the non-infected patient:

> *Scoles* v. *Mercy Health Corp.*, 1994 W. L. 686623 (Universal precautions failed to satisfactorily reduce the threat of transmission posed by an HIV-infected surgeon, due to the fatal nature of HIV), which settled before appeals.
>
> *Mauro* v. *Borgess Medical Center*, 886 F. Supp. 1349. (W.D. Mich. 1995). (HIV infected surgical technician posed a direct threat because of the "real possibility of transmission, however small, and because the consequence of transmission is invariably death").

APPENDIX C

THE INTERESTS OF THE HEALTH OF THE DENTAL ASSISTANTS/HYGIENISTS WHO PERFORM INVASIVE DENTAL PROCEDURES FOR KNOWN PATIENTS WITH HIV INFECTIOUS DISEASE

Scientific findings prove that the hospital operating room can be the best invasive treatment environment not only for many patients with HIV infectious disease, but also for the dental assistants/hygienists.

Since 1994, medical advances have been made which significantly extend the lives of patients with HIV infectious disease if administered in a proper and timely manner. These modern drug "cocktails" have also proven effective in stopping the infection for someone who has been exposed to the virus. These drugs are called post-exposure prophylaxis drugs (PEP drugs). The Centers for Disease Control reported in 1996 that if an injured dentist or dental assistant/hygienist receives PEP drugs within one hour after an occupational HIV exposure, the possibility that he/she will contract HIV infectious disease is reduced by about 79%. "PEP should be initiated promptly, preferably within 1-2 hours postexposure."[4] This urgent need for the timely administration of PEP drugs following an exposure to HIV infectious disease is still recommended by the CDC.[5] If a dental healthcare worker is occupationally exposed to HIV infectious disease, it is best that he/she is exposed while working in a hospital operating room due to instant availability of the PEP drugs.

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[4] *Morbidity and Mortality Weekly Report* by the Centers for Disease Control and Prevention. Vol. 45, No. 22. June 7, 1996, page 470.

[5] *Morbidity and Mortality Weekly Report*. Vol. 47, No RR-7. May 15, 1998, page 1.

Dr. David K. Henderson of the National Institutes of Health, made the following statement in a recent issue of the *Journal of the American Medical Association*:

> As promptly as feasible, the health care worker should report the exposure to her/his institution's occupational medical department...If prophylaxis is elected, the first dose should be administered as promptly as possible. *We strive to administer the first dose within 1 hour of the exposure* (emphasis added).[6]

The downside of the anti-HIV drugs is that drug-resistant human immuno-deficient viruses (HIV) have come into existence. The mutated viruses void any extension of life realized by utilizing the modern treatment "cocktails." This has further significantly raised the anxiety level of many healthcare workers who perform invasive procedures for known patients with HIV infectious disease.

Dr. Bragdon's decision in 1994 was supported by 1994 scientific data, has since been further endorsed by post-1994 scientific data (including the discovery and use of PEP drugs), and was non-discriminatory and fair to all concerned. He demonstrated prudent clinical judgment when suggesting to Ms. Abbott that she have her invasive dental healthcare treatment performed in the hospital operating room.

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[6] Henderson, Dr. David K. "Postexposure Chemoprophylaxis for Occupational Exposures to the Human Immunodeficiency Virus." *Journal of the American Medical Association.* Vol. 281, No.10. 10 March 1999, page 931.

APPENDIX D

THE INTERESTS OF THE HEALTH OF THE PRACTICING DENTIST WHO PERFORMS INVASIVE DENTAL PROCEDURES FOR KNOWN PATIENTS WITH HIV INFECTIOUS DISEASE

1) Common knowledge concerning the hospital operating room option when planning invasive dental healthcare procedures for a patient with HIV infectious disease:

Historically, the hospital was designed to prevent the spread of infectious disease from infected patients to non-infected people—healthcare workers included.

It is common knowledge that the hospital operating room offers a sanctuary from the distractions of the everyday fast-paced dental office. Physicians have traditionally opted for the operating room instead of their private offices for a variety of reasons, including the less stressful and safer environment.

Without the stress and distractions of the dental office to contend with, the dentist and dental assistant are better able to concentrate on the treatment procedure and take the necessary time to reduce potential accidental occupational exposures to HIV infectious disease.

In 1994, Dr. Bragdon believed and the American Association of Forensic Dentists and Clinical Research Associates concurred, that the hospital operating room could help healthcare workers provide better invasive dental healthcare services for patients with HIV infectious disease in a safer manner, minimizing needle-stick and other occupational exposures to HIV.

2) Scientific findings prove that the hospital operating room is a reasonable option for the dentist in which to invasively treat known patients with HIV infectious disease.

The American Dental Association states there is no evidence of HIV transmission from infected patients to dentists. This overstates the case. There is indeed evidence of HIV transmission, but no scientific proof of it. This is because so few dentists have been enrolled in the requisite prospective studies (essential for documentation) that only 19 exposures have been documented.[7] Since only one exposure in 300 is thought to transmit HIV, it is not surprising that no transmissions to dentists have been documented, including the seven possible transmissions to dentists that are listed in the CDC's HIV/AIDS Surveillance data. OSHA has reported the potential for HIV transmission due to a single contaminated needle stick is 3 to 4 per 1,000.[8] Dr. David Bell, the CDC's Director for the HIV Infectious Programs, has reported that the risk of getting HIV infectious disease from some hollow-bore needle stick injuries is 5%.[9] Sanford Kuvin, M.S., M.D., D.T.M.&H, Dr. Bragdon's expert witness, testified that a reasonable estimate of the risk that a dentist will accidentally contract HIV infectious disease when giving a single intra-oral injection (shot of local anesthesia) to an HIV-infected patient is 1 in 66,667. Dr. Kuvin further testified that if a dentist gives an average of one intra-oral injection each working day to a patient with HIV infectious disease for thirty years, the dentist has a 1 in

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[7] "Percutaneous Exposures to HIV Infected Blood." *Journal of the American Dental Association.* Vol. 126. September 1995, page 1237.

[8] "Occupational Exposure to Bloodborne Pathogens: Final Rule." *Federal Register.* Vol.. 56, No. 235. Dec. 6, 1991, page 64090.

[9] *Amicus* Brief of Defendant/Appellant Randon Bragdon, D.M.D., to the United States Court of Appeals for the First District, on Remand from the United States Supreme Court, pages 5 and 6.

10 risk of accidentally contracting HIV infectious disease during the lifetime of the practice.[10] The CDC has reviewed several studies and concluded that dentists traditionally have a high rate of needle stick injuries (2-15 injuries per month per dentist).[11]

In 1994, injury with HIV infected instruments (e.g. needle sticks) was considered significant by many members of the medical community. "It is becoming clear that the risk of occupational HIV exposure may be significant."[12]

3) The courts should not consider additional prudent precautions, which can protect the health of dentists, as discriminating against known patients with HIV infectious disease.

There are many other additional prudent precautions, besides the hospital operating room option, which dentists have available. Some of these are: using only sharp single-ended instead of double-ended instruments, or double gloves instead of single, or treating these patients at the start or end of a work day instead of during high traffic hours in their dental office, or suspending the use of aerosol producing instruments, or the utilization of high volume evacuation under laminar flow conditions with hood or full face protection and remote air source for the operator.

Dentists are concerned that these prudent additional precautions may be considered by the courts to be discriminatory. Therefore, they can be fearful of legal

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[10] *Ibid.*

[11] Ciesielski, C. "Transmission of HIV in a Dental Practice." *Annals of Internal Medicine.* Vol. 116, No. 10. May 15, 1992, pages 798-805.

[12] Hermann, Richard C. "Risks of HIV Exposure to Medical Students and Health Care Personnel." *Journal of the American Medical Association.* Vol. 264, No. 9. 1990, page 1187.

consequences of implementing them. This affects the health interests of many dentists who desire to use these other prudent additional precautions. The "direct threat' issue of this case directly relates to the use of these other additional prudent precautions.

Because of concern for the non-efficacy of universal precautions, the lack of sound scientific determinations concerning "degree of risk," and the concern for needle-stick and other occupational exposures when invasively treating patients with HIV infectious disease, Dr. Bragdon was reasonable when he suggested to Ms. Abbott that she have her invasive dental healthcare procedures performed in the hospital operating room. Post-exposure prophylaxis drug science further validates Dr. Bragdon's, or any dentist's, decision to recommend the additional precaution of the operating room for the invasive treatment of known patients with HIV infectious disease.

ADDENDUM

PROFESSIONAL RESUMES OF ADDITIONAL AMICI

1. Congressman Charles Norwood, D.D.S., represents Georgia's 10th Congressional District. He has served as President of both the Georgia Dental Association and the Eastern District Dental Society, was elected as a Delegate to the American Dental Association in 1984-85, and was founder of the Augusta Dental Disaster Society. He remains a member of the American Dental Association, the Georgia Academy of Dental Society, the Augusta Dental Society, is an Honorable Fellow of the Georgia Dental Association, and a Fellow of both the International College of Dentistry and the American College of Dentistry Sciences.

2. Congressman Tom Coburn, M.D., represents Oklahoma's 2nd Congressional District in the U.S. House of Representatives. A practicing physician, Coburn is a Member of the Committee on Commerce where he serves as the Vice-Chairman of the subcommittee on Health & Environment. He graduated the University of Oklahoma Medical School in 1983. Coburn is a resident of Muskogee, Oklahoma, where he specializes in family medicine and obstetrics. Dr. Coburn and his associates serve more than 10,000 patients.

3. The American Association of Forensic Dentists (AAFD) is an international association of dental, medical, human biology and law enforcement professionals organized in 1978 to promote scientific and sound treatment of diseases, human identification, epidemiology and legal matters related to the field of dentistry. The AAFD's membership rolls number approximately 3,000. The American Association of Forensic Dentists previously

submitted an *Amicus Curiae* brief in support of Dr. Bragdon to the United States Court of Appeals for the First Circuit.

4. The Association of American Physicians and Surgeons, Inc. is a national association of over 5,000 physicians of all specialties, founded in 1943 to preserve and promote the practice of private medicine in the tradition of Hippocrates.

5. Dentists for Preservation of Professional Judgement is an organization founded in April, 1997, and composed mostly of American Dental Association members and other healthcare related individuals. Dentists for Preservation of Professional Judgement previously submitted an *Amicus Curiae* brief in support of Dr. Bragdon to the United States Court of Appeals for the First Circuit.

6. E.J. Neiburger, D.D.S., is Director of the Center for Dental AIDS Research, Past Scientific Chairman of the Illinois State Dental Society, Past Chairman of the St. Therese Hospital Dental Staff, Vice President of the American Association of Forensic Dentists, and Editor of the Journal of the American Association of Forensic Dentists since 1979.

7. Cary Savitch, M.D., is an assistant clinical professor of medicine at UCLA, and a Fellow in the American College of Physicians. He has practiced medicine since 1977. Throughout his medical career, he has specialized in the study and treatment of infectious diseases. In particular, from his infectious disease specialty training at the San Francisco General Hospital in 1979-1980 up to

the present, he has studied both HIV and AIDS and has treated and consulted on hundreds of patients with AIDS and/or HIV. Dr. Savitch previously submitted an *Amicus Curiae* brief in support of Dr. Bragdon to the United States Court of Appeals for the First Circuit.

8. Stephen D. Carter, D.D.S., is a member of the American Dental Association, Georgia Dental Association, the Northern District Dental Society of Georgia, and the Pierre Fouchard Academy. He is a dentist-researcher who holds three patents related to infection-control related research: for Basic research in heat independent autoclaving utilizing agents in conjunction with increased pressure and ultrasonic transient cavitational collapse; Basic research in compression/ supersaturation/explosive decompression of microbes for germicidal effect; and for Basic research and development of the Kleen system of disinfecting writing instruments in the healthcare setting. He has had numerous articles published on infection control related subjects. He has 32 years experience as a practicing dentist. He is an Honorable Fellow of the Georgia Dental Association.

9. John L. Kennedy, D.D.S., is a member of the American Dental Association, the New Mexico Dental Association, and the Laguna Beach, Ca. Dental Study and Think Tank Group. He was a contributing Editor to "Dental Economics" magazine from 1989-1997. He was President of the Baylor College of Dentistry Alumni Organization for 1996-97 and a Honors Graduate of Baylor University College of Dentistry 1964. He is a Consultant for Mercer International and has had 31 years experience in the private practice of Dentistry.

10. Charles E. Smith, D.M.D. is a Member fo the American Board of Dental Examiners, a Member of the Alabama Board of Dental Examiners, a Past President of the Third District Dental Society, a component of the Alabama Dental Association which is a constituent of the American Dental Association. He has had 32 years experience as a practicing General Dentist.

11. Brian E. Eifert M.D. is a member of the Roentgen Ray Society, the Association of American Physicians and Surgeons, the Society of Nuclear Medicine, and is President-Elect of the Mississippi Society of Nuclear Medicine. He is a practicing radiologist.

12. William J. Dubose, B.S, D.M.D. is a past President of the Alabama Dental Association, a constituent of the American Dental Association. He is a Fellow in the American College of Dentistry, a Fellow in the International College of Dentistry, and a Master in the Academy of General Dentistry. He has had 40 years experience as a practicing General Dentist.

13. Robert J. Gherardi, B.S., D.M.D., FAGD, FACD, FADI, is a member of the American Dental Association and served the Association as: Vice-Chairman of the Council on Dental Therapeutics in 1991, member of the Reference Committee on Scientific Matters in 1992, and Delegate from New Mexico from 1990-1998. He is a member of the Academy of Dentistry International, American College of Dentists, the New Mexico Board of Dental Health, the National Academy of General Dentistry, the New Mexico Dental Association, the Albuquerque District Dental Society, and is a past President of the New Mexico Academy of General Dentistry. He received the following fellowships: the Academy of General Dentistry in 1987, the Academy of Dentistry International in 1993, and the American

College of Dentists in 1996. He was listed in 1995 in "Who's Who" in American Dentistry. He has had 19 years experience in the private practice of general dentistry.

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No. 98-1536

In The

Supreme Court of the United States

October Term, 1998

RANDON BRAGDON, D.M.D.,

Petitioner,

v.

SIDNEY ABBOTT, et al.,

Respondents.

On Petition For A Writ Of Certiorari To The United States Court Of Appeals For The First Circuit

BRIEF OF THE *AMICUS CURIAE* CLINICAL RESEARCH ASSOCIATES IN SUPPORT OF RANDON BRAGDON, D.M.D., PETITIONER

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STATEMENT OF INTEREST OF *AMICUS CURIAE*

Clinical Research Associates hereby submits this brief as *amicus curiae* in support of Dr. Randon Bragdon, D.M.D.[1] The brief is submitted by the written consent of all parties to the suit.

Clinical Research Associates, known worldwide as CRA, was initiated over 23 years ago by dental clinicians to test the efficacy and clinical usefulness of products they use in dental treatments. CRA is a unique non-profit research organization comprised of 45 on-site basic scientists, engineers, and support staff, plus over 400 dental clinicians located in 18 countries who volunteer their time and expertise to field test products within their practice environments. CRA's activities have been compared to Consumers' Union and the Consumers' Report publication, although CRA works only with products used in dental treatments. CRA performs about 20,000 product evaluations each year, working with about 650 different products secured from the worldwide market. Results of CRA's work are published monthly in nine languages and circulated to 72 countries.

Since its inception, CRA has worked actively with infection control products, including over 60 different face-mask brands, 36,000 gloves, 20 different types and styles of protective clothing, over 150 different environmental surface disinfectant formulations, 25 instrument immersion disinfectants, 15 commercial heat sterilizers,

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[1] Pursuant to Supreme Court Rule 37.6, this brief was prepared entirely by counsel for CRA. No monetary compensation was received from any other person or entity.

12 different designs of air purification equipment, and over 40 different products sold for treatment of dental unit waterlines. CRA is the only place in the world where all types of infection control products used in dentistry receive extensive efficacy testing routinely.

CRA's mission and only purpose is to test products used in dental treatments for efficacy and clinical usefulness, and to communicate to the dental community all results.

SUMMARY OF ARGUMENT

Throughout the proceedings of this case in the lower courts, the following key issue has failed to receive consideration: Efficacy of the "universal precautions" concept, claimed by experts to protect clinicians and patients against significant risk of transmission of infectious diseases, is *flawed seriously* by the fact that it is totally dependent on the efficacy of *products* used to implement it (face-masks, operating gloves, disinfectants, sterilizers, etc.). CRA's lab has tested thousands of products used for infection control to support the universal precautions concept over the past 23 years, and has demonstrated clearly and repeatedly that most of these products available worldwide fail to meet their intended purposes. *Failure of infection control products that support the universal precautions concept virtually negates the concept.* Although there may be a visual appearance of protection, if masks fail to filter properly, operating gloves leak, and disinfectants and sterilizers fail to kill as expected, this places both clinicians and patients at significant risk every time

infectious organisms are present. Furthermore, efficacy of *all* the products, efficacious or not, needed to protect clinicians and patients from infectious disease transmission is easily compromised by clinical mistakes and accidents, which can occur unexpectedly at any time and inflict a lifetime of serious ill health and/or untimely death. (*See* reference # 1, pages App. 1-4).[2]

ARGUMENT

THE UNIVERSAL PRECAUTIONS CONCEPT DOES NOT ALLEVIATE SIGNIFICANT RISK TO CAREGIVERS DURING TREATMENT OF INFECTIOUS PATIENTS BECAUSE THE PRODUCTS NECESSARY TO IMPLEMENT THE CONCEPT OFTEN FAIL THEIR INTENDED PURPOSE

Because microbes are invisible, clinicians cannot determine by themselves the efficacy of the infection control products they use. They have no way to test for face-mask filtration capacity, operating glove leakage, and disinfectant and sterilizer kill. Ideally, efficacy of these critical products should be confirmed before each patient treatment, but no tests exist anywhere that provide this capability. CRA has demonstrated that product cost cannot be used as an indicator of product efficacy. So this leaves clinicians totally at the mercy of the product manufacturer's integrity. All the above factors open

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[2] All articles referenced herein are reproduced in the Appendix to this brief, noted by reference number and page number.

opportunities for extensive deception and fraud in product promotional claims.

Today none of the agencies originally entrusted with safeguarding the public against non-efficacious products used in healthcare such as the EPA, FDA, CDC and American Dental Association, actually *test* the products they certify or recommend. Although doubts about disinfectant efficacy have been pointed out by the General Accounting Office (GAO), (*See* reference # 2, pages App. 5-13), lack of government funding for a central testing lab allows the problem to continue unabated.

Today, after many years without non-biased confirmatory testing there now exists a large number of products promoted as protective against transmission of infectious microbes that, in fact, do not fulfill that purpose. Clinicians purchase and use these products routinely, hoping they are protected. The authors of the universal precautions concept and the experts who have testified in this case that no precautions should be taken by caregivers beyond those prescribed by the universal precautions concept have all *assumed* that the products that support the concept perform as expected. This means the products are assumed to block microbes or kill microbes before they enter the clinician's body.

CRA's work demonstrates that these assumptions are ill founded. In actuality, every time a person with an infectious disease is treated, the clinician plays Russian roulette with his own health, and his life, as well as his attending staff and subsequent patients.

This brief is supported by data from studies performed by CRA and others, included in the appendix

hereto, which clearly demonstrate that this *blind faith* in the protection afforded by infection control products (universal precautions) is *not justified.*

Of critical importance in the instant case, the data demonstrate the protective measures commonly referred to as universal precautions do *not* provide the protection claimed by the authorities and experts referenced in the testimonies cited in this case. Unfortunately, the experts whose testimony was presented by Respondents in the proceedings before the First Circuit on remand[3] are not microbiologists actually involved in testing efficacy of infection control products.

It appears the experts based their testimonies on the assumption that large numbers of healthcare providers are not believed to have become HIV positive from work-related exposures. This does not mean there is not significant risk to caregivers using the universal precautions concept. There are cases on record where experts have been frustrated and perplexed because serious transmissions occurred in the presence of all the proper universal precautions. These cases point clearly to the fact that universal precautions strategies can, and do, fail to protect both clinicians and patients. (*See* references # 3 & 4 on pages App. 14-40).

Every breach does not elicit an infection, as the referenced paper illustrates. Although the hepatitis B infected

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[3] This testimony is contained in CDC's 1987 *Recommendations for Prevention of HIV Transmission in Health-Care Settings,* 36 MMWR No. 2S (Aug. 21, 1987), as referenced in Dr. Bragdon's brief at pages 4-5.

surgeon caused disease in 18 patients, another 124 he treated did not contract the disease. This is because a whole set of circumstances generally must coincide at the moment of organism transfer in order for the infection to result in subsequent disease. These circumstances are thought to include, but are not limited to, such factors as: (1) virulence of the microorganism at the time of transfer, (2) numbers of the microorganism transferred, and (3) the immune status of the recipient at the time of transfer. Hence, in every case, *chance* plays a part in whether or not infection will escalate to disease. Indeed, the word "disease" is defined as "failure of the *adaptive mechanisms* of an organism to counteract adequately the stimuli or stresses to which it is subject, resulting in disturbance in the function or structure of any part . . . " (Blakiston's Pocket Medical Dictionary) (4th ed. 1976) (emphasis added). Therefore, it needs to be understood from the beginning that determination of "significant risk" is more than a numbers game of how many infected people constitute "significance." It is, more importantly, a determination of the importance of each human life suddenly affected by chance when a face-mask or glove leaks infectious materials, or a disinfectant or sterilizer fails to kill critical microbes, or a needle or other sharp instrument penetrates barriers. In truth, determination of significant risk pits the life of the infected patient against the life of the uninfected caregiver, and the court is charged with the responsibility to choose which is to be favored.

CRA submits the following data as evidence that the products used to implement the universal precautions concept cannot be relied upon to alleviate the significant

risk always present when clinicians treat patients with infectious diseases:

1. **Face-Mask Efficacy (*See* reference # 5 on pages App. 41-56).**

When 42 different face-masks and 2 face shields used by dental clinicians in the U.S. were tested for ability to block aerosolized bacteria, only 22 of the 42, or about half (52%), passed this test. In further tests with the 42 masks, when blue dye was aerosolized to simulate microbe laden aerosols generated during dental procedures, and a human subject within a sealed chamber breathed the dye laden aerosols, *all* the masks (100%) showed significant leakage through to the mask interior, and *only one* of the 42 masks was designed in such a way that it could be adapted at its border to fit securely enough to block dye penetration at this critical interface. We have since found that all face-masks sold worldwide to dental clinicians become overloaded quickly (within 10 to 30 minutes) and allow material to penetrate to the mask interior, and only two masks are designed that prevent ingress of materials at the periphery.

This penetration of debris aerosolized from the patient's oral cavity is highly significant because it brings infectious organisms from another person's body into direct contact with the lips, nostrils, and skin, and into the respiratory passages. This affords the possibility for *direct contact of critical mucous membrane tissues* with infectious material.

Current mask designs do not provide any way for clinicians to determine when leak-through begins or the

amount present. Therefore, as masks entrap and fill up with aerosolized material, and leak to the mask interior, the mask becomes a source of, rather than a barrier to, aerosolized infectious microorganisms.

Dental handpieces and their cutting drills used to perform the tooth drilling emit large amounts of air and water under pressure while rotating at speeds up to 250,000 to 300,000 rpm. These instruments cause massive aerosolization of patients' oral tissues. Research confirms that about 50% of the aerosols generated during dental procedures are three µm and smaller in size. Such particles remain suspended at the end of treatment for long time periods, creating risk even after treatment of infectious patients is completed. Furthermore, three µm and smaller aerosols can penetrate the human respiratory system all the way to the alveoli of the lower respiratory tract, which greatly increases infectivity. Although the experts in this case claimed that they know of no cases where HIV has been transmitted by aerosols, in dental applications the possibility of such a transmission cannot be denied. It is known that HIV particles can be present in saliva. It is known that saliva is aerosolized during use of dental handpieces, ultrasonic scalers, air polishers, and in many other less obvious dental treatments. It is known that face-masks do not present an impenetrable barrier. These facts lead to the conclusion that risk is present, and transmission of HIV to alveolar macrophages and other key cells within the lungs is possible during dental procedures.

CRA submits that the referenced data show face-masks do not provide the protection expected by dental clinicians from infectious saliva, blood, pus, tooth debris

and other materials aerosolized during dental treatment, and they leave clinicians subjected to significant risk of disease transmission while creating the illusion of protection.

2. Latex Examination Glove Efficacy – (*See* references # 6-10, pages App. 57-84).

Over its 23 years of operation, CRA has tested over 36,000 clinical operating gloves made of latex, vinyl, nitrile, and other non-latex materials. If we consider just the gloves made of latex, since these are the most used, CRA has tested 84 different brands manufactured in the U.S., Malaysia, Taiwan, India, China, and Thailand. If we combine the pinhole data[4] for latex gloves from just the test results published in the enclosed references, and do not include any other reports, we find an overall prevalence of 6.75% pinholes in the glove brands tested. (*See* references # 6-9, pages App. 57-77.) Most pinholes are not visible to the naked eye, but can be disclosed by filling the glove with air and submerging it in water, by filling the glove with water and looking for seepage of the water to the external after a few minutes, by viewing the glove under a scanning electron microscope, or by rubbing gloved hands of humans with dye and then looking for dye spots on the hands of the person after degloving.

Most dentists treat about 15 patients per day and work about 240 days per year. The 6.75% pinholes found

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[4] A pinhole is defined as a small breach in the integrity of the glove material generally resulting from manufacturing problems.

by CRA allows *the average dentist to use about 486 gloves in a year that have holes in them* that permit the patient debris, which the dentist wears gloves to avoid, to leak through the gloves and onto their hands. Using the 4% pinhole maximum allowed by the FDA for latex examination gloves, a dentist would use 288 gloves in a work year that leaked.

All of CRA's glove integrity test data above is the result of testing new, unused latex examination gloves. Logically, clinical use stresses latex gloves, and causes one to suspect an increasing number of breaches in this barrier as use time and work challenges increase. Tests performed at Johns Hopkins University School of Nursing (*See* reference # 10, pages App. 78-84) showed a volume increase in fluid leaked by 240 latex gloves tested of 0.08 µl in new unused gloves vs. 2.20 µl in gloves subjected to simulated clinical procedures. This represents more than a 27-fold increase in material leaked due to usage. So not only must clinicians beware of leakage of new, unused gloves, but they must expect significantly more leakage as they move through routine procedures. And it is self-evident that latex gloves are easily penetrated by sharp instruments. It is also evident that it is not necessary to be exposed to a large quantity of infected blood to become HIV positive following an accident (*See* reference # 1, pages App. 1-4).

What number of glove leaks constitutes a "significant risk," within the meaning of 42 U.S.C. § 1282(b)(3)? CRA submits that the referenced data show latex examination gloves do not provide the protection expected by dental clinicians from infectious saliva, blood, pus, tooth debris, soft tissue, and other materials handled during dental

treatment, and they leave clinicians subjected to significant risk of disease transmission while creating the illusion of protection.

3. Environmental Surface Disinfectants (*See* references # 11-15, pages App. 85-160).

Environmental surface disinfectants are liquids used on a vast variety of objects in homes, offices, hospitals, restaurants, hotel rooms, and dental operatories. They are used on all types of surfaces ranging from toilet seats and bowls, kitchen sinks and counters, and walls and floors everywhere, to objects in dental operatories and hospital operating rooms.

The wide variety of applications and uses has inspired the creation of a large number of products, which unfortunately do not perform equally well. Yet to most people, any container labeled "disinfectant" is presumed to kill everything, instantly and completely. This perception represents a gross misunderstanding of the chemistry of disinfectants and the physiology of microorganisms, and can cause serious mistakes to be made by those who select the disinfectants to be used in clinical treatment areas, exposing the clinical personnel to significant risk.

Although the potential of contaminated environmental surfaces to transmit diseases has been questioned, confirmation of the fact that organisms are not rendered non-viable immediately upon leaving the body has always made it impossible to rule out environmental surfaces as fomites. In addition, experiments designed

specifically to investigate this point have repeatedly demonstrated transmission of both infection and disease. For example, in experiments using human hands contaminated with hepatitis A virus, it was shown clearly that the virus was transferred from hand to hand, from hands to objects, and from objects to hands (*See* reference # 11, pages App. 85-108). In other experiments, healthy human volunteers were infected with colds by handling tiles and cup handles contaminated by the investigators with rhinovirus (*See* reference # 12, pages App. 109-118). While CRA is unaware of similar experiments using HIV, experiments by competent researchers experienced in handling HIV reported viable virus still present after five minutes of exposure to a 10% dilution of household bleach. (*See* reference # 13, pages App. 119-124).

This indicates that HIV is relatively resistant, at least to the 1:10 bleach, which has been recommended as the environmental surface disinfectant of choice by the CDC for many years.

Since clinicians perceive all disinfectants as equally effective, they shop for formulations that are cosmetically pleasing (agreeable fragrance, does not corrode or stain, visually pleasing packaging, etc.), and never stop to consider microbial kill. This is done under the misplaced assumption that consumers are protected from ineffective disinfectants by EPA and FDA regulations. Although these agencies have the authority to control which disinfectants are sold within the U.S., neither performs confirmatory tests for efficacy and both lack a simple and clear communication system to inform consumers about what the disinfectant can and cannot do. These problems have resulted in use of disinfectants that do not kill

properly and leave viable organisms intact and able to infect people in most of the medical and dental facilities throughout the U.S.

Of 39 environmental surface disinfectants commonly used by dental clinicians and tested by CRA in 1988-89, only three, or 8% passed all the tests. (*See* reference # 14, pages App. 125-158). This means 92% of 39 products could not kill a clinically representative, resistant virus (poliovirus) and bacteria (the tuberculosis bacteria, *Mycobacterium bovis*) in both the absence and presence of human blood.

Five years later after packaging, ownership, and formulation changes by many disinfectant manufacturers, CRA revisited this work and added some new formulations. So far, 59 formulations have been tested in this second round and only seven have passed all the tests, (*See* reference # 15, pages App. 159-160), which means 88% still fail to kill under clinically relevant conditions.

Of the six disinfectant *active ingredients* used worldwide, only two provide broad spectrum disinfection, and even these must be used at specific dilutions and/or in specific combinations with certain other chemicals, or they also fail to kill. Ethyl alcohol at about 70% by weight and household or institutional bleach diluted no more than 1:2 were the only formulations that provided reliable kill, regardless of the test method, test organism, or contact time used, both in the absence and presence of fresh human whole blood.

CRA submits that the referenced data show that most environmental surface disinfectants do not provide the microorganism kill expected by dental clinicians, and

they leave clinicians subjected to significant risk of disease transmission while creating the illusion of protection.

4. Instrument Immersion Disinfectants (*See* references # 16-17, pages App. 161-170).

Formulations of instrument immersion disinfectants can differ from environmental surface disinfectants, so they are considered separately here. Glutaraldehyde (ideally at dilutions of about 2% or 3%) has been the worldwide standard for instrument immersion for the past ten-plus years. It should not be used for environmental surfaces because it emits fumes that can be toxic when it is used liberally on countertops in unventilated areas, it can elicit skin sensitivities, and it generally has a slow kill of the tuberculosis bacteria that would extend surface contact times beyond all practicality. However, as an instrument immersion disinfectant its characteristics of reliable broad spectrum kill, even in the presence of gross amounts of bodily contaminants such as blood, feces, tissue, etc., make it a very unique and valuable clinical product.

However, even glutaraldehyde disinfectants are not foolproof. In 1991, when CRA tested 12 different formulations (sold under 12 different brand names), water dilutions of the disinfectants suggested on the labels proved to be a significant problem. (*See* references # 16-17, pages App. 161-170). Of 15 recommended dilutions, 14 (93%) required *more time* than stated on labels to kill poliovirus and the TB bacteria. Five required *hours* longer than the times stated, with two requiring *five hours* longer than

times stated on the labels. Since 1991 when CRA performed this work, FDA has attempted to correct this problem and has eliminated the worst discrepancies, but some problems still exist with kill-time label claims on some products. Ironically, if clinicians are willing to allow any amount of time, up to six hours, for disinfection to take place, CRA data showed all the products tested eventually killed the test organisms adequately.

CRA submits that even the most potent disinfectants, known as "high level disinfectants", now regulated under the auspices of FDA, have not provided protection expected by clinicians, leaving them subjected to significant potential for disease transmission, while creating the illusion of protection.

5. Sterilization Equipment (*See* reference # 18, pages App. 171-184).

Heat sterilization equipment is one area of infection control products where governmental control (FDA clearance) has been very strict, and proof of a substantial margin of safety has been required. However, the decision to "grandfather" some sterilizers sold before 1976, and still sold today, presents problems because they have not been subjected to the same performance criteria.

However, the biggest problem in the dental sterilizer market is dental drill sterilization. It has been shown that dental drills generally require a little more time to render them sterile than the conventional hand instruments, such as mouth mirrors and "picks" used by dentists. Therefore, FDA requires a separate submission and confirmatory data for dental drill sterilization.

Unfortunately, dental clinicians are totally ignorant of this requirement. Clinicians purchase and use their sterilizers for all instruments, including dental drills. There is no realization by clinicians that the dental drills may require a longer time to be rendered sterile.

In the summer of 1998, a researcher shocked the dental community by reporting that several models of chemical vapor sterilizers did not render dental drills sterile. (*See* reference # 18, pages App. 171-184). He demonstrated lack of sterility by recovering viable test spores within the dental drills. CRA then examined the U.S. dental sterilizer market and found that five of ten popular dental sterilizers (50%) had not received 510K clearance for handpiece sterilization, even though manufacturers and distributors of these sterilizers knew that dental clinicians were likely to use the sterilizers to treat dental drills.

Therefore, sterilization times for dental drills processed in the five sterilizers yet to be cleared are unknown. The clinical risk related to this fact can only be surmised.

Because of the significant risk that exists under the universal precautions protocol, at a minimum Dr. Bragdon should have been allowed to present evidence such as that contained herein to the finder of fact. The First Circuit erred in affirming the summary judgment in favor of Respondents.

CONCLUSION

CRA has addressed the term "significant risk" as it is used in the Americans with Disabilities Act related to dental clinical treatment of HIV positive and AIDS patients. Data and comments presented are meant to address testimonies of experts in this case who declared that no protective measures beyond those known as "universal precautions" are necessary to secure dental clinicians against transmissions during treatment. CRA has submitted a body of data generated by our lab and others to show that the products necessary to implement the universal precautions concept (face-masks, latex gloves, disinfectants, and sterilizers) are defective even when fresh, new and unused. CRA submits that when the products fail to block or kill as expected, the universal precautions concept is negated, and clinicians who operate thereunder are exposed to significant risk. The situation is analogous to requiring every infantry person to carry into battle a rifle that is known to have a defective trigger that allows the gun to be fired sometimes, as expected, but jams sometimes without warning. Would anyone disagree that the soldiers were at risk of getting shot and maybe killed when their gun jammed and the enemy got off the first shot?

To place people infected with a virus transmitted by their blood and other body fluids into routine mainstream healthcare, and then disallow any special precautions simply invites catastrophic incidents from time to time, without any way to predict exactly when a caregiver will become infected. The nature of viral transmission is generally haphazard unless the virus is particularly virulent and/or the immunity pool is nonexistent.

Therefore, all exposure incidents may not lead to infection and disease, and the courts may be tempted to play a numbers game to determine significance of the risk. The unfortunate part of this approach is that the infection is exquisitely significant to the caregiver infected, and possibly also to the index patient involved in the transmission. The point is, we now have *two* infected individuals instead of just the original one. If we wanted to play a numbers game, we could say we just doubled the original number of infected people, and doubling of a number is considered a significant increase to most people.

CRA's data indicate there *is* significant risk, not overcome by universal precautions, every time HIV positive patients receive treatment where bleeding tissue and use of sharp instruments is involved.

Is it safer for dentists to treat HIV positive patients in a hospital? The answer to this question is both yes and no. No, it probably does not make a difference to the safety of the infected patient. YES, it could be safer to the uninfected clinicians because, in the event of a clinical accident, within the hospital they can receive *immediate* prophylactic treatment. Prophylactic treatment involves administration of drugs not readily available just anywhere. Ideally, post-exposure drugs should be administered immediately after exposure to allow the best chance of effectiveness. In suburbs and rural areas, after accident, clinicians may have to lose precious time traveling to centers where these drugs are available. In some cases, the travel could involve a delay of many hours, which could diminish chances of a positive outcome.

Analysis of Dr. Bragdon's petition should *focus on control of infectious diseases*. While unfounded discrimination is improper under the ADA and within our society, when there exists a significant threat to the life of a caregiver the law should not prevent the caregiver from taking all reasonable precautions. The data presented herein show that significant risk exists every time clinicians treat an infected patient using the defective defenses of the universal precautions protocol. This risk is escalated exponentially when an accident or mistake occurs that causes further compromise of universal precautions products, which are highly questionable from the onset.

Theoretically, the products that support the universal precautions concept should provide the protection sought, in the absence of any accidents that compromise them. But, in actuality, the infection control products provide no more than a *show* to reassure patients that the personnel treating them are aware of, and concerned about, transmission of diseases, and to reassure clinicians by placing a shield or barrier between their body and infectious materials. But any thinking clinician realizes the protection is weak and minimal and easily penetrated in a split second of an unfortunate move. Most clinicians and patients (and, for that matter, the experts who testified in this case), are *unaware* of the flaws in masks, gloves, disinfectants, and sterilizers, which we have reported here. Based on the evidence herein, to say or imply that universal precautions products are enough to remove significant risk of viral transmission during treatment of HIV positive patients is wishful thinking at best,

and reckless disregard for the health and lives of caregivers and other patients at worst.

Respectfully submitted,

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REFERENCE NO. 1

The Boston Globe – Tuesday, April 6, 1999

A nurse hopes her infection will spur action

By Richard A. Knox
Globe Staff

Nurse Karen Daley was shoving a used hypodermic needle into a disposal box at the Brigham and Women's Hospital emergency room last July when she felt a sharp pain. A used needle inside the box was pointing upward, and it stuck her gloved finger.

Months later she got the awful news: The accidental needle-stick injury had infected her with the viruses that cause AIDS and hepatitis C, a serious and often fatal liver disease.

US health-care workers report about 600,000 accidental injuries from needles and other "sharps" each year, resulting in about 1,000 serious infections. What made Daley's case unique is that she is president of the Massachusetts Nurses Association.

Ironically, the association was already preparing legislation that would require hospitals to use the best available needle stick prevention technology.

To further the cause, the 46-year-old nurse plans to reveal her infection publicly today in Beacon Hill testimony.

"She's one amazing person," said Gloria Craven, the association's lobbyist. "She's saying 'How can we turn this into something good?'"

Daley declined to speak about her situation before her testimony, but colleagues say she is coping with stoicism and determination. She reportedly suffers serious fatigue from her infections.

Through June of last year, the US Centers for Disease Control recorded 54 cases of documented HIV infection among health-care workers and 133 possible cases. Nurses led the list of workers with occupationally acquired HIV infection.

It is rare for victims of accidental needle sticks to become infected with both hepatitis C and the human immunodeficiency virus (HIV), which causes AIDS. Both are potentially fatal, and hepatitis C is the leading cause of liver failure requiring transplantation.

The dual infection complicates Daley's treatment, a nurse colleague said, and makes it difficult to predict her course.

Although details of Daley's injury were unavailable yesterday, it may have been impossible to identify the patient who was the source of her infections. The disposal box presumably contained needles for a variety of patients treated that day.

One of Daley's colleagues called her injury "100 percent preventable" by such means as better-designed disposal boxes, more frequent emptying of such receptacles, and needles that are covered by a retractable plastic

sheath except at the moment when they are in contact with a patient's skin.

"Overall the health-care industry has not seen fit to provide the devices that would make the work environment safer for nurses and others," said Evelyn Bain, an occupational safety and health specialist at the nurses association.

The costs of safer technology are far outweighed, Bain said, by the costs of investigating needle-stick incidents and providing prophylactic drugs costing $1,300 to $1,800 per case, not to mention the future treatment costs and human suffering if a health-care worker becomes infected. "One local hospital that reduced needle-stick injuries by 50 percent with a prevention program still recorded 101 injuries in 1997," she added. "That facility spent $141,000 to follow the injured workers."

The proposed legislation, which will be considered by the Legislatures's Joint Health Care Committee today, would set up a mechanism within the state health department requiring health-care facilities to employ best-available prevention strategies.

"Facilities would have to do only three things: purchase the appropriate equipment; look at how to prevent injury; and when an injury occurs, document it and keep a log so we can use it for quality improvement," Craven said.

So far Oregon is the only state to pass such a law, she said.

Judy Glasser, a spokeswoman for the Massachusetts Hospital Association, said hospitals generally support the

proposal. "We believe it moves in the right direction, and we believe we should do everything we can to protect our employees and caregivers," Glasser said.

Staff members at the nurses association said they learned only about a month ago about Daley's infections, which the administration of prophylactic drugs had failed to prevent. "Most of us haven't stopped crying yet," one said.

Daley's career may not be over, but her 25 years of direct patient care may be at an end because of the risk she may pose to patients. About 85 percent of individuals infected with hepatitis C virus become chronic carriers, and so far no treatment has been shown to eliminate HIV from the tissues of infected individuals.

REFERENCE NO. 2

United States General Accounting Office
Report to Congressional Requesters
August 1990

DISINFECTANTS: EPA Lacks Assurance They Work

GAO/RCED-90-139

Executive Summary

Purpose

Disinfectants – about a $1 billion per year market – are used to kill germs on inanimate surfaces and objects in hospitals, schools, restaurants, and homes. Because users cannot see whether disinfectants kill bacteria, fungi, and viruses, the use of ineffective disinfectants poses a threat to public health and wastes consumer dollars.

Mounting concerns about whether hospital and household disinfectants work as claimed and the adequacy of the Environmental Protection Agency's (EPA) disinfectants program led the House Committee on Government Operations to request that GAO review EPA's regulation of the efficacy of disinfectants.

Background

Under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), EPA generally must register (license) pesticide products, including disinfectants, before they

are marketed. EPA may register a pesticide product only if EPA determines that it is effective, when used as claimed, without causing an unreasonable risk to health or the environment. For most pesticides, EPA allows the marketplace to regulate product performance (efficacy) because users can see whether the pesticide is effective against the target pest. However, registrants of disinfectants intended to protect public health must submit efficacy data to substantiate each product performance claim and use.

Until 1982, EPA conducted limited preregistration confirmatory and post-registration enforcement tests on disinfectants at its laboratory facilities in Beltsville, Maryland. EPA discontinued disinfectant testing in 1982 primarily because of budget constraints. Currently, EPA relies on its review of registrant-submitted efficacy data to register disinfectants. As of September 1, 1989, about 4,100 disinfectants for public-health use were registered with EPA, representing about 18 percent of approximately 23,000 registered pesticide products.

Results in Brief

EPA does not know whether disinfectants kill the germs claimed on product labels for four reasons. First, although the validity of

methods and performance standards used to assess the efficacy of disinfectants has been the source of scientific controversy for over a decade, EPA does not independently test disinfectants before registering them and lacks criteria to assess the validity of registrant-proposed test methods and modifications. Second, EPA has made little progress in resolving these controversies because of budget constraints and inadequate research management. Third, EPA lacks sufficient internal controls to ensure the quality and integrity of the data that registrants submit on disinfectant efficacy. Fourth, EPA lacks an enforcement strategy to ensure that, once registered, disinfectants sold and distributed in the marketplace work as claimed.

The extent to which ineffective disinfectants are marketed is unknown. Although the scientific controversies cloud the issue somewhat, evidence from EPA, the states, and others suggests that up to 20 percent of disinfectants on the market may be ineffective.

Principal Findings

Validity of Test Methods

EPA lacks assurance that the test methods and performance standards

used by registrants to substantiate disinfectant efficacy claims are valid. EPA primarily relies on standard-setting organizations, such as the Association of Official Analytical Chemists (AOAC) and the industry itself to develop test methods and performance standards. However, these methods and standards have been embroiled in scientific controversies for over a decade. For example, scientists have debated whether the AOAC Use-Dilution Method, the most widely used test method, is reproducible, accurate, and precise, and whether the performance standard (pass/fail criterion) established by EPA is valid. Although EPA believes that the existing methods and standards are acceptable for registering and enforcing disinfectant efficacy claims, the controversies have impaired the credibility of the disinfectant program. An ad hoc industry/state group recently has developed a test method to replace the AOAC Use-Dilution Method and is expected to present the results of its research to the AOAC in September 1990. EPA officials believe that the new method is reproducible and reliable and will consider whether to require that disinfectants be retested using it after AOAC considers it for adoption.

EPA has contributed to the controversies by accepting test methods

and modifications without criteria and independent laboratory data for evaluating their validity. For example, EPA accepts three different test methods to demonstrate that disinfectants kill tuberculosis bacteria. At least one product tested under two of the methods produced substantially different results. Although EPA has registered the product on the basis of one of the methods, EPA lacks the laboratory information needed to explain the differences in results between the methods.

Although EPA has been aware of the scientific controversies for years, it has made little progress in resolving them because of problems in conducting needed research. EPA's 6-year, $384,000, cooperative agreements with the University of North Carolina did not fulfill EPA's research objectives to improve disinfectant efficacy methods because EPA inadequately managed the agreements. EPA has also made little progress in conducting additional research because of budget constraints. In April 1990, EPA announced that it would spend $600,000 for research on certain disinfectant efficacy methods and estimated an additional $1.2 million will be needed.

Controls Over Quality and Integrity of Data

To ensure the quality and integrity of registrant-submitted disinfectant data, EPA reviews the data prior to registration and performs laboratory inspections and data audits. GAO, however, found internal control weaknesses in these programs. For example, EPA has not inspected the majority of labs that have performed disinfectant efficacy studies. In fact, EPA was aware of only 12 of the 92 labs that had performed these studies. Although these programs need to be improved they, in themselves, are not an adequate substitute for a preregistration program to selectively test disinfectant efficacy by an independent laboratory. Data reviewers, lab inspectors, and data auditors generally cannot identify cases in which registrants have selectively submitted data indicating that their disinfectants work because they generally do not observe the tests in progress and no physical evidence remains from the tests conducted.

Monitoring and Enforcement of Marketed Disinfectants

EPA's registration process by itself cannot provide assurance that disinfectants are effective because registrants could market ineffective batches, either intentionally or inadvertently, after registering them.

However, EPA does not enforce the efficacy claims of disinfectants on the market. EPA discontinued its limited enforcement testing program in 1982 primarily because of budget constraints. Since 1982, EPA has looked to the states, user groups, and the industry to enforce efficacy claims. However, GAO found few states and no users monitoring disinfectant efficacy because of cost concerns. Only five states test disinfectants for efficacy, and these states have limited programs. Moreover, EPA lacks a strategy to channel complaints about potentially ineffective disinfectants from the states, user groups, and the industry and to take appropriate enforcement action against disinfectants found to be ineffective. Although EPA needs to resolve the scientific controversies that surround disinfectant efficacy test methods and performance standards, these controversies should not prevent EPA from developing an enforcement strategy, in conjunction with the states, user groups and industry, to ensure that marketed disinfectants work as claimed. Public health and consumer welfare may be comprised without such assurance.

Recommendations

GAO is making recommendations to the Administrator, EPA, to correct deficiencies and restore credibility

in the disinfectant program, including (1) developing a plan to resolve the scientific controversies that surround disinfectant efficacy test methods and performance standards; (2) developing and publishing a policy that establishes criteria for evaluating the validity of new test methods and modifications, including criteria for determining when independent laboratory data are needed for validation; (3) improving internal controls over its current programs to ensure the quality and integrity of registrant-submitted efficacy data and conducting preregistration tests to selectively verify registrant claims; (4) establishing an enforcement strategy in conjunction with the states, user groups, and industry to ensure that marketed disinfectants work as claimed; and (5) preparing a cost-benefit analysis of alternatives for a laboratory facility to research and test the efficacy of disinfectants, including the option of charging fees to register disinfectants to help finance such a facility.

Agency Comments

GAO did not obtain official agency comments on this report. GAO did, however, discuss the factual content of the report with EPA officials and has included their comments where appropriate. EPA officials generally agreed with the accuracy of the facts

but believed that, as presented, the report could be misread and suggested changes for presenting the facts. GAO made some revisions to the report on the basis of EPA's comments. GAO believes that the report is a fair and accurate presentation of the issues.

REFERENCE NO. 3

New York Times, March 22, 1994

Investigating a Medical Maze: Virus Transmission in Surgery

Hepatitis B was apparently spread by a doctor, but how?

By LAWRENCE K. ALTMAN, M.D.

In a cluster of cases that has mystified medical detectives, 18 patients who underwent heart surgery at two hospitals associated with the University of California at Los Angeles over a 10-month period in 1991 and 1992 developed hepatitis B several weeks after their operations.

The sleuths believe they know who the carrier of the virus was: a surgeon. But only after more than a year and a half of investigation do they have a clue as to how the patients became infected.

By reviewing medical records, interviewing doctors and patients and testing their blood, epidemiologist traced the cluster in July 1992 to a surgeon-in-training who had not been immunized against the viral liver infection. Over the 10 months, the surgeon, who has not been identified, had been involved in operations on 142 patients at both the U.C.L.A Medical Center and the Wadsworth Veterans Administration Hospital. Of these, 18, 13 percent developed hepatitis B infection. Of at least 155 patients operated on by other surgeons at the two hospitals, none developed hepatitis B.

A team of experts from the Federal Centers for Disease Control and Prevention in Atlanta, U.C.L.A. and the Los Angeles County Health Department tested as many of the 18 patients as they could, and found that in every one the virus was identical to that of the surgeon. The team reported its findings at a meeting in Atlanta last month.

The evidence was so compelling that the doctor stopped working as a surgeon as soon as the cluster was detected. He is now working in the operating room of an unidentified hospital and not performing surgical procedures, according to Dr. James Cherry, a U.C.L.A. hospital epidemiologist and an author of the report.

The doctor had presumably been infected by a patient, whom the detectives were not able to identify. It takes about six months from the time of exposure before symptoms, including fatigue and jaundice develop. In the doctor's case, this occurred about halfway through a period in which the patients were infected. The epidemiologist suspect he infected some patients while incubating the disease.

The doctor was tested, found to have hepatitis B and was permitted by the hospital to return to surgery after about two weeks, even though he was now a carrier of the virus.

The sleuths had solved one dimension of the outbreak. Still, a mystery remained: how did the surgeon transmit the virus to so many patients?

He did not recall having stuck himself with a needle or sharp instrument. If such a puncture tore his glove, a

few drops of virus-contaminated-blood could have leaked into a patient's body. This is also the way the surgeon himself might have become infected, though by the time he developed symptoms six months later, he had no recollection of any such incidents.

But as Dr. David M. Bell, an expert on hospital infections for the Centers for Disease Control and Prevention and a co-chairman of the meeting pointed out, the surgeon would have had to injure himself scores of times to infect so many patients, Colleagues had not noticed that the surgeon's operating-room techniques were flawed or sloppy.

After spending months pursuing many leads without conclusively identifying a specific mechanism for transmitting the virus, the epidemiologist decided to pick up on one of the surgeon's own suggestions.

"He mentioned that when he operates, on occasion his fingers would become irritated, and he speculated that perhaps the pressure of tying sutures might have something to do with the transmission" of the virus, said Dr. Rafael Harpaz, a CDC epidemiologist who headed the team, in an interview.

So the surgeon cooperated in a simulation of his surgical technique. In a sort of surgical marathon, he tied knots for an hour, far more than he would do in a real operation. When he finished the simulation, his fingers were bruised. The inside of each glove was washed, and the washings were sent to a laboratory. To the investigators' surprise, virus was found in the washings, offering a possible explanation of how the surgeon might have transmitted the virus to his patients: it might have

escaped through tiny holes in his surgical gloves, the result either of manufacturing imperfections or tears from use.

Surgeons often note minor bruising from suturing and the pressure of holding instruments, but they generally do not equate such irritations with the seriousness of an accidental glove puncture from a needle, sharp instrument or piece of bone.

It takes just a few drops of hepatitis B-contaminated blood to be infectious. Studies show that the risk of transmitting hepatitis B from a needle stick is about 100 times as great as the risk of transmitting the AIDS virus.

Transmission of hepatitis from an infected patient to a health care worker is not unusual. But Dr. Harpaz said the Los Angeles cluster is one of just seven reported worldwide since 1987 of transmission in the other direction, affecting some 44 patients altogether. Ability to transmit hepatitis B correlates strongly with the presence of the so-called e antigen in the blood. The antigen often remains but can disappear over time.

In several of the clusters, health officials were not able to determine how the hepatitis virus had been transmitted to a patient. They assumed that the doctor was sloppy and violated standard infection-control practices. But Dr. Bell of the CDC said that there was little scientific data to support this assumption and that "we need to re-examine it in light of the findings" from the Los Angeles cluster.

In none of the other cases were simulations and laboratory studies used to study how transmission might

have taken place. Dr. Harpaz said, while Dr. Bell and other experts cautioned against generalizing from the Los Angeles cluster until additional studies are done, he said that if the finding is confirmed, he hoped it could lead to improving gloves to help prevent transmission.

Although the spread of hepatitis B has never been linked to sweat, participants at the meeting suggested this might be a mode of transmission. If so, they speculated that contaminated sweat could leak through a hole in surgical gloves into a patient's body, thus posing a previously unrecognized risk.

Like HIV, the virus that causes AIDS, the hepatitis B virus is transmitted by blood and through sexual contact. But unlike HIV, hepatitis B can be prevented by a vaccine.

Dr. Craig Shapiro of the Centers for Disease Control, who is an author of the Los Angeles study, said that until recent years, an estimated 250 health care workers a year developed hepatitis B and eventually died from it. The figure now is about 100 a year, in part due to a recommendation in 1991 from the Federal Occupational Safety and Health Administration that surgeons and other health care workers be immunized to prevent hepatitis B infection.

One puzzle in the Los Angeles outbreaks is why the surgeon had refused the vaccine, which would have protected himself and his patients. Because health officials have not identified him, there is no way to ask him why he had not been vaccinated to protect himself and his patients. But Dr. Cherry of U.C.L.A. said the surgeon had been offered the vaccine at a hospital he had worked at before coming to U.C.L.A.

Dr. Quentin R. Stiles, a retired chest surgeon in Los Angeles who is a spokesman for the American College of Surgeons, said "A lot of surgeons are like that, mostly older ones," in declining to take the very immunization that they recommend to their patients. He characterized these older surgeons as "a very reluctant bunch" in changing their ways.

Because of their exposure to blood, surgeons run a particular occupational risk of getting hepatitis B. Yet the college has not conducted a survey to determine how many surgeons have taken the series of three hepatitis B shots. The vaccine has been available since 1981.

Dr. Stiles recently told a group of surgeons that they face a greater risk from hepatitis B than from HIV. When he asked how many in the audience had been immunized against hepatitis B, fewer than half said they had, and most of them were younger surgeons. Immunizing surgeons against hepatitis B "is the kind of thing we ought to be insisting upon," Dr. Stiles said.

In 1992, the Association of American Medical Colleges in Washington recommended that all medical students be immunized against hepatitis B before their first contact with patients and with blood products and human tissue. Immunization is of no benefit after infection with the virus. The association has not conducted a survey to determine compliance with the recommendation.

While the hospital's decision to allow the doctor to keep on operating even after he was found to be infected with the virus may seem unusual, it is in compliance with Federal guidelines. These call for local committees to

decide whether an infected surgeon can continue operating or performing procedures in which patients could be exposed to infected blood. In deciding, the committee evaluates a number of factors that might bear on the risk of transmission, like the skill of the worker and the kinds of procedures that the individual performs.

Health officials are aware that nothing less than the doctor's career is at stake in the decision.

The policy of the Los Angeles County Health Department, according to Dr. Shirley L. Fannin, an official of the department, is to allow infected doctors to continue working. But the doctor is informed that if one case of transmission is detected, limitations will be imposed on his practice.

REFERENCE NO. 4

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TRANSMISSION OF HEPATITIS B VIRUS TO MULTIPLE PATIENTS FROM A SURGEON WITHOUT EVIDENCE OF INADEQUATE INFECTION CONTROL

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Abstract *Background.* Although about 1 percent of surgeons are infected with hepatitis B virus (HBV), transmission from surgeons to patients is thought to be uncommon. In July 1992, a 47-year-old woman became ill with acute hepatitis B after undergoing a thymectomy in which a thoracic-surgery resident who had had acute hepatitis B six months earlier assisted.

Methods. To determine whether the surgeon transmitted HBV to this patient and others, we conducted chart review, interviews, and serologic testing of thoracic-surgery patients at the two hospitals where the surgeon worked from July 1991 to July 1992. Hepatitis B surface antigen (HBsAg) subtypes and DNA sequences from the surgeon and from infected patients were determined.

Results. Of 144 susceptible patients in whose surgery the infected surgeon participated, 19 had evidence of recent HBV infection (13 percent). One of the hospitals

was selected for additional study, and none of the 124 susceptible patients of the other thoracic surgeons at this hospital had evidence of recent HBV infection (relative risk, ∞; 95 percent confidence interval, 4.7 to ∞). No evidence was found for any common source of HBV other than the infected surgeon. The HBsAg subtype and the partial HBV DNA sequences from the surgeon were identical to those in the infected patients. Transmission of the infection was associated with cardiac transplantation (relative risk, 4.9; 95 percent confidence interval, 1.5 to 15.5) but not with other surgical procedures. The surgeon was positive for hepatitis B e antigen and had a high serum HBV DNA concentration (15 ng per milliliter). Our investigations identified no deficiencies in the surgeon's infection-control practices.

Conclusions. In this outbreak there was surgeon-to-patient HBV transmission despite apparent compliance with recommended infection-control practices. We could not identify any specific events that led to transmission. (N Engl J Med 1996;334:549-54). ©1996, Massachusetts Medical Society.

APPROXIMATELY 24 million operations are performed annually in U.S. hospitals by an estimated 133,000 surgeons.[1] The Centers for Disease Control and Prevention (CDC) estimate that 1900 U.S. surgeons are chronically infected with hepatitis B virus (HBV), but reports of surgeon-to-patient transmission of the virus are uncommon.[2] Transmission of HBV to patients has been associated with health care workers with highly infectious disease who were positive for hepatitis B e antigen (HBeAg) and has generally involved breaches in standard infection-control practices, although correction of these

deficiencies has not always prevented additional instances of transmission.[3-6] We report an outbreak of HBV infection associated with an HBV-infected thoracic-surgery resident and suggest potential mechanisms of transmission.

METHODS

In July 1992, a 47-year-old woman without identified risk factors became ill with acute hepatitis B four months after undergoing a thymectomy in which a thoracic-surgery resident participated. This surgeon was found to be susceptible to HBV on testing in December 1989 before completing a general-surgery residency elsewhere. He began the thoracic-surgery residency program in July 1991 after a year of research. He was offered the hepatitis B vaccine but never received it. In January 1992, he became fatigued, and in February he had jaundice with detectable hepatitis B surface antigen (HBsAg) and IgM antibody to hepatitis B core antigen (anti-HBc). He withdrew from surgical duty until March 1992, when his symptoms resolved, and he returned to practicing surgery having had no additional tests for HBsAg or HBeAg. He was still positive for HBsAg and HBeAg in July 1992, when the index patient was identified, and was relieved of surgical duties pending an investigation.

To determine whether other patients were infected with HBV, we obtained blood specimens in September 1992 from patients operated on by the surgeon during the study period, July 1991 to July 1992. The surgeon worked at two hospitals during this period, referred to here as Hospital A and Hospital B. For specimens collected

within six months after surgery, we asked susceptible patients to provide second specimens six months or more after surgery to permit the detection of later seroconversion. Chart reviews and interviews of patients or their parents were conducted with standardized forms; demographic data and information about surgical characteristics, prior HBV infection, and community risk factors for infection were recorded. Sexual and household contacts of infected patients were tested to exclude them as sources of transmission. Patients were defined as having acute HBV infection (case patients) if they were IgM anti-HBc-positive or if they were seronegative within the year before surgery or on initial testing and positive for HBsAg or anti-HBc on final testing.

Retrospective Cohort Studies

A retrospective cohort study was conducted at Hospital A to determine whether the surgeon had transmitted HBV to the index patient and possibly others. We compared the risks of infection among patients he operated on and among patients who underwent thoracic surgery without his participation from November 1991 to July 1992. Patients who had thoracic surgery without the surgeon's participation were contacted by letter and follow-up telephone call and asked to provide demographic information and serum for evaluation. This retrospective cohort study included all patients who underwent thoracic surgery during the months when the surgeon was not working at Hospital A and every third patient of other thoracic surgeons during the months when he was working there.

A second retrospective cohort study evaluated risk factors for infection among the patients the surgeon operated on at Hospital A, where data were more complete. The study period was the same as that of the first retrospective cohort study. Data regarding characteristics of operations and surgical procedures were collected with standardized forms.

Additional information was collected by informally interviewing the surgeon, operating-room personnel (thoracic-surgery fellows, attending physicians, anesthesiologists, scrub nurses, and perfusionists), and nurses in the intensive care unit. The work schedules of nurses, phlebotomists, and respiratory therapists were reviewed for possible opportunities for transmission by circulating hospital personnel. The vaccination records and the results of serologic tests for HBV of operating-room personnel were reviewed, as were records of transfusions in patients. Testing was conducted at the CDC for HBsAg and antibody to HBsAg (anti-HBs) by radioimmunoassay and for anti-HBc and IgM anti-HBc by enzyme immunoassay (Abbott Laboratories, North Chicago, Ill.). In several instances, samples were tested at local clinical laboratories. Samples with detectable HBsAg were analyzed for HBsAg subtype by enzyme immunoassay with monoclonal antibodies.[7]

Analysis of Serum Samples

Serum from the surgeon, from the infected patients, and from an unrelated, acutely and chronically infected convenience sample of controls from the state in which the outbreak occurred were subjected to amplification by

the polymerase chain reaction (PCR) to detect HBV DNA. Twenty microliters of serum was digested with proteinase K solution for one hour at 65°C, followed by phenol-chloroform extraction and alcohol precipitation. HBV1858 (5'ACTGTTCA-AGCCTCCAAGCTG3'), HBV2437 (5'TTGAGATCTTCTGCGACGCGGC3'), and 5 units of Taq polymerase were added to the precipitate for amplification (30 cycles consisting of denaturation at 95°C for 30 seconds, annealing at 55°C for 30 seconds, and extension at 72°C for 45 seconds). The samples were purified and the sequence of 160 bases in the core region was determined with use of HBV1858P[8] or the ABI automated sequencer and dye terminators (Applied Biosystems, Foster City, Calif.). The sequences were analyzed with the Pileup program, which performs progressive, pairwise comparisons and plots the results in a dendogram to indicate similarity of sequences.[9]

The HBV DNA concentration in the surgeon's serum was determined by dot blot hybridization[10] and by PCR end-point dilution. Ten-fold serum dilutions were amplified by PCR and evaluated by agarosegel electrophoresis. The threshold of HBV DNA detectability was compared with that of similarly diluted serum containing 100 million chimpanzee-infectious particles per milliliter.

Statistical Analysis

The relative risks and 95 percent confidence intervals were calculated with the use of Epi Info[11]; the associations between exposures and infection were assessed by univariate and stratified analysis; and the significance of differences in proportions was assessed by the chi-square

test with the Mantel-Haenszel correction for independent samples or with Fisher's exact test.

RESULTS

Identification of Cases

The surgeon operated on 239 patients (162 at Hospital A and 77 at Hospital B) from July 1, 1991, through July 16, 1992. Twenty-eight patients died before the investigation; none had recognized evidence of hepatitis. Of the remaining 211 patients, 184 (87 percent) were available for initial serologic testing, with 170 (81 percent) tested six months or more after surgery. Of these 170, 11 reported prior HBV infection or hepatitis B vaccination and had markers consistent with their histories; they were excluded from the analysis. Nineteen patients had evidence of acute HBV infection, as indicated by the presence of IgM anti-HBc or by anti-HBc seroconversion. Fifteen additional patients had HBV serologic markers but were negative for IgM anti-HBc and had no evidence indicating the presence or absence of seroconversion; seven of these patients had other risk factors for HBV infection. For purposes of analysis, these 15 patients were assumed to have been infected before surgery. The overall attack rate was therefore 13 percent (19 of 144) among susceptible patients available for follow-up.

The 19 case patients with acute HBV infection ranged in age from 14 months to 83 years (median, 51 years). Six (32 percent) had symptoms of acute hepatitis, one of whom required hospitalization. Three of the remaining patients died of other causes. Chronic HBV infection developed in 9 of the 16 surviving case patients (56

percent); 3 of these 9 had been receiving immunosuppressive therapy, and another was two years of age.

All 19 case patients or their parents reported no other risk factors for HBV infection. Sexual and household contacts of all but three patients underwent HBV testing; none were HBsAg-positive. Of the 19 case patients, 15 had had surgery at Hospital A and 4 at Hospital B. The procedures included coronary-artery bypass surgery (eight), orthotopic heart transplantation (four), repair of congenital heart defects (four), valve replacement (one), thymectomy (one), and open-lung biopsy (one). The procedures occurred throughout the study period without apparent clustering in time (Fig. 1), even after we controlled for the number of susceptible patients undergoing surgery each month (data not shown).

Determining the Source of the Outbreak

We conducted a retrospective cohort study to determine whether patients of other surgeons at Hospital A had acute HBV infection: a sample of 280 of 510 such patients who underwent surgery from November 1991 through June 1992 was selected for evaluation, including all patients who underwent thoracic surgery at Hospital A while the HBV-infected surgeon was not working there and every third patient of other thoracic surgeons at Hospital A while he was working there. Of these patients, 259 were alive; 124 consented to be tested, were determined to be susceptible at the time of surgery, and had six-month follow-up data available (Table 1). None of the 124 had evidence of recent HBV infection. This result contrasts with the 15 who had such evidence (14 percent)

among the 106 patients who were operated on by the surgeon at Hospital A (Table 1) (relative risk, ∞; 95 percent confidence interval, 4.7 to ∞).

The two cohorts did not differ significantly according to age, sex, race, or the distribution of procedures (data not shown), although the average duration of surgery was longer for patients operated on by the infected surgeon than for patients operated on by other surgeons (5.4 vs. 4.5 hours; $P<0.001$). When patients undergoing thoracic surgery who had evidence of prior, but not recent, infection were included in the analysis as case patients, the difference in infection rates for the two cohorts remained significant: 24 of the 115 patients operated on by the infected surgeon were HBV-infected (21 percent), in contrast with 7 of the 131 patients operated on by other surgeons (5.3 percent) (relative risk, 3.9; 95 percent confidence interval, 1.8 to 8.7).

Opportunities for transmission from other nosocomial exposures were investigated. Twelve of 15 case patients at Hospital A (80 percent) had received blood transfusions, all from different donors. No other surgeon, anesthesiologist, nurse, phlebotomist, or respiratory therapist had documentation of HBV infection, and none treated more than seven case patients. Transmission to patients occurred at both Hospital A and Hospital B; the surgeon was the only common factor at the two hospitals.

The HBsAg subtype from the surgeon and from 13 of the 19 case patients at both hospitals for whom subtyping could be performed was adw2. We amplified HBV DNA from the surgeon, 9 case patients, and 19 unrelated community controls (7 with acute infection and 12 with

chronic infection). The sequences from the surgeon and the case patients were identical. The sequences from all but 4 of the 19 community controls were different from each other and from the sequence from the surgeon (Fig. 2).

Evaluation of Surgery-Related Risk Factors for HBV Infection

To identify risk factors for HBV infection, we conducted a retrospective cohort analysis of the patients the surgeon operated on at Hospital A (Table 2). The infection rates did not differ according to sex or age. The rate was higher among whites than in other racial groups. The infection rate was higher among patients who received blood products during surgery or who had surgery lasting 5.5 hours or longer, but these differences were not statistically significant. The surgeon's patients underwent a variety of surgical procedures, but the infection rate was increased only among patients who underwent cardiac transplantation (relative risk, 4.9; 95 percent confidence interval, 1.5 to 15.5). This association remained significant when the duration of the procedure and the use of blood products were controlled for (data not shown). The infection rates were not associated with emergency (as opposed to elective) surgery, the use of a perfusion pump or cell saver, the specific operating room, or prior sternotomy. The analysis of these associations was unchanged by the inclusion of the 15 patients with serologic markers of HBV infection but without evidence of recent seroconversion (data not shown).

Additional Fact-Finding

The surgeon reported no risk factors for HBV infection and was unaware of any percutaneous exposure to blood from HBV-infected patients. He indistinctly recalled only one or two needle sticks during the period under investigation, and he reported no injuries from sternal wires or other sharp objects. He reported that he always handled sharp objects with an instrument and did not blindly palpate suture needles. Although the surgeon reportedly often applied hemostatic material to sternal incisions with his gloved hands rather than with the protection of a sponge (the usual practice of other surgeons at the two hospitals), he recalled no glove punctures from this procedure. He performed no invasive procedures on case patients in the intensive care unit or the recovery room.

Other surgical personnel attested to the surgeon's good technique. He was left-handed, which sometimes interfered with the passing of instruments or simultaneous suturing by more than one surgeon. He did not use double gloves, but after contracting hepatitis B he modified his behavior by frequently changing gloves during operations. All surgical staff members, including the surgeon, reported that blood was routinely present on their hands when they removed their gloves after an operation, whether or not visible tears were present in the gloves and regardless of the type of gloves used.

In previous years, the surgeon had had a skin irritation that resolved after he changed to the routine use of hypoallergenic latex gloves. In addition, he had periodic

pain over the radial side of the index fingers that he attributed to shear forces from tying sutures.

The serum HBV DNA concentration in the surgeon just after the index patient was identified was 15 ng per milliliter. The serum was estimated by semiquantitative PCR to contain 1 billion infectious particles per milliliter.

Discussion

The infected thoracic surgeon whom we studied transmitted HBV to at least 19 patients during surgery. No patients undergoing procedures performed by other thoracic surgeons had evidence of recent infection. The timing of the infections and the absence of other identified sources of infection among the case patients were consistent with transmission from the surgeon during surgery. The infections occurred at two different hospitals without common equipment or staff members other than the surgeon. The presence of HBsAg subtype adw2 in the surgeon and in the 13 cases patients in whom the subtype of the antigen could be determined is unlikely to have occurred by chance alone.[12] The DNA sequences of the HBV core region from the surgeon and from all 9 case patients who could be evaluated were identical and were different from that of all but 4 of the 19 community isolates.

Although reporting of HBV infection is not complete, both outbreaks and sporadic transmission of HBV from surgeon to patient appear to be uncommon. Evidence that the risk is low is limited and includes retrospective studies involving patients of infected health care workers,[13-16] two case-control studies of patients with

acute hepatitis B that found no association between disease and surgical history[17] (and unpublished data), and the relatively small number of reported outbreaks of HBV given the estimated pool of infected surgeons.

Outbreaks provide information about specific mechanisms of transmission of HBV from surgeon to patient. Since the early 1970s, 29 such clusters have been reported worldwide,[3,5,18-24] including 9 involving thoracic surgeons.[5,19,22-24] Data from these outbreaks indicate an increased risk of HBV transmission from HBeAg-positive surgeons and during particularly invasive procedures.[5,21,25] Transmission during many of these outbreaks was presumed to be caused by deficiencies in infection-control measures. Although this outbreak involved a high attack rate, our investigation did not identify any breaches in infection-control practices, despite an extensive search for potential modes of transmission. Unreported or unrecalled percutaneous exposures by the surgeon or operating-room staff are unlikely to explain such a high rate of transmission.

Although this is the first reported outbreak involving a thoracic surgeon in the United States, four such outbreaks have been reported in the United Kingdom during the past decade.[5,24] We found no specific features characteristic of thoracic surgery that were associated with transmission. Surgical fields are generally well visualized during thoracic surgery, and blind needle palpation is not often practiced. Thoracic surgery is, however, inherently highly invasive and of long duration, and these features have been linked to percutaneous exposure,[26-28] glove failure,[29-31] and HBV transmission.[21] Indeed, whether caused by the duration of surgery or by specific factors

such as the closure of sternotomy incisions, frequent glove punctures during thoracic surgery have been reported.[26,28,31,32] In this outbreak, there was no association of HBV infection with the duration of surgery or the use of blood products (a possible indication of the invasiveness of a procedure); two case patients, in fact, underwent brief procedures requiring no blood products (a thymectomy and an open-lung biopsy). We found no associations between HBV transmission and specific procedures, with the exception of cardiac transplantation, although in relative terms these were not long or complex procedures. Perhaps the minimal infectious inoculum of HBV is lower for patients receiving immunosuppressive therapy. Some surgeons have suggested that closure of the median sternotomy incision is associated with injury, although data to support this assertion are inconclusive.[31,32] In our study, the surgeon's technique of applying hemostatic material to the sternal incision without a sponge may have caused injuries that were not apparent. However, one case patient underwent an open-lung biopsy that did not involve a median sternotomy.

This outbreak may have been related more closely to factors unique to the surgeon than to factors inherent in thoracic surgery: indeed, lung biopsy is a procedure with little resemblance to most other thoracic surgical procedures. Although HBeAg-positive persons almost always have highly infectious disease, the surgeon had an especially high concentration of HBV DNA during the outbreak, which may have contributed to aq high risk of transmission. The surgeon's technical skills were apparently not a factor, since operating-room personnel did not

recall that he had frequent needle sticks. The hand irritation experienced by the surgeon in previous years had resolved with the use of hypoallergenic latex gloves, and there was no evidence that the surgeon had dermatitis during the outbreak. Hypoallergenic gloves are subject to the same quality standards as standard surgical gloves.

The surgeon had pain over his index fingers during prolonged suturing. Other surgeons have described similar experiences to us; we are unaware of any studies addressing this phenomenon. While participating in a one-hour simulation of suture tying,[33] the surgeon acquired paper-cut-like lesions on his fingers, and HgsAg and HBV DNA were isolated from washings of his hands. Such lesions, combined with the failure of his gloves, may have allowed contamination of patients with HBV. Although gloves frequently have leaks during surgery,[27,28,31,34,35] they nonetheless appear to be fairly effective barriers against certain infections, even when leaks are present.[36] Although there is increasing evidence that double gloves can prevent exposure of surgeons to blood during surgery,[35] there is no evidence regarding the effectiveness of double gloves in protecting patients from blood-borne infections. Furthermore, advisory groups and professional organizations have not generally recommended the use of double gloves by surgeons. Additional studies are needed to assess the validity and generalizability of the suture-tying simulation and to define the role of gloves in preventing the intraoperative transmission of HBV.

This outbreak has had tragic consequences for the case patients, their families, and the surgeon, who has left surgical practice indefinitely. The entire episode could

have been prevented had the surgeon received the hepatitis B vaccine.

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REFERENCE NO. 5

GENERAL DENTISTRY/NOVEMBER-DECEMBER 1991

Efficiency of 42 brands of face masks and 2 face shields in preventing inhalation of airborne debris

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Use of face masks by dental clinicians has increased as a result of the AIDS epidemic.[1] In response to clinicians' concerns about infectious disease transmission, the American Dental Association (ADA) and Centers for Disease Control (CDC) issued recommendations for use of preventive measures, including face masks, during dental treatment.[2,3] The use of face masks is now required by the Occupational Safety and Health Administration (OSHA).[4,5] However, none of these agencies provided information on relative merits of different types of facial barriers, which might give one the impression that all facial barriers are equally effective. While this may be true for blockage of large particles and splatter, dentists also must protect themselves from aerosols that are generated during dental procedures and remain airborne following patient treatment.[6-11]

In clinical studies characterizing aerosols by particle size, Pollok et al. showed that about half of such particles were 3 µm or smaller.[6] Aerosol particles of this size have been shown to be particularly critical. In studies of 26 nonsmokers, Chan et al. demonstrated that particles about 3µm in size have maximum deposition in the alveoli.[12] According to Day et al., this deep penetration

into the respiratory tract increases the infectivity of microorganisms.[13] Hence, face masks for dental use should block particles down to at least 3 μm in size.

Studies of toxicological warfare have confirmed that airborne organisms can cause infections in man.[14-15] To determine whether tooth-cutting procedures could lead to aerosols containing oral pathogens in numbers sufficient to infect humans, Belting et al. cut cavity preparations on tuberculosis patients with positive sputum and cultured air samples following treatment.[8] They showed conclusively that oral tuberculosis organisms become airborne in numbers sufficient to infect clinicians. At present, the use of high-filtration facial barriers with close peripheral fit is the best known way to minimize inhalation and infections from this type of airborne bacteria.

Clinicians need to know the differences among the various brands of facial barriers on the market. This study compares the efficacy of various face masks and shields in blocking airborne debris.

Materials and methods

The test protocol specified four evaluations:

(1) Three samples of all face masks studied were tested in vitro.

(2) Selected face masks worn by clinicians were tested to determine clinical effectiveness.

(3) Selected face masks were worn in high humidity, then tested to determine the high humidity's effect.

(4) Finally, a dye tracer test was performed on selected face masks and shields to determine importance of peripheral fit.

Collection of masks and shields

Brand names of face masks sold to dental clinicians were identified, and identical masks sold under different brand names were eliminated. However, when confidentiality agreements prohibited identification of manufacturers, similar masks were tested, as clinicians at times have no way to confirm manufacturers and often must identify face masks by brand name only. Face shields were selected on the basis of market popularity and amount of facial coverage. The table lists brand names of 42 face masks and 2 face shields evaluated.

Bacterial filtration efficiency testing

Evaluation of 42 brands of face masks – Three unused samples of each of 42 brands of face masks were coded and sent for bacterial filtration efficiency (BFE) testing at a commercial laboratory that routinely performs this standardized procedure developed by Nicholes (Fig.1).[16] A 24-hour culture of *Staphylococcus aureus* clinical isolate, strain Utah 15, at a concentration of approximately 10^4 colony forming units (cfu) per milliliter, was used in the following manner:

(1) The culture suspension was nebulized (Chicago nebulizer*) to create a cloud with a mean particle size of 3.0 μm.

(2) The cloud was drawn through the test mask at a rate of 1 ft^3/min. and onto a 6-stage

sampler (Andersen sampler†) where particles were separated by size onto one of 6 Petri plates containing soybean casein digest agar (Fig. 2a and 2b).

(3) The plates were incubated at 37°C for 48 hours; colony forming units were counted and probable hit values were computed according to Andersen's method.[17]

To determine the consistency of delivery for the challenge aerosol, control values were established through sampling without a filter. Acceptable limits for this aerosol density were between 1,700 and 2,700 cfu/ft.^{3} To determine proper functioning of the test system, a reference filter with a known BFE was tested. Challenge aerosol sampling and reference material evaluation were conducted for every 8 to 10 test samples.

The following formula was used to calculate filtration efficiency of all test masks:

$$\text{Percent BFE} = \frac{C - F}{C} \times 100,$$

where C (control) represents the number of particles sampled when no mask was used an F (filtered) represents the number of bacterial particles that passed through the test mask. Mean BFE percentages were computed for each set of three identical masks. One-way analysis of variance (AOV) and the Bonferroni multiple comparison procedure were used to determine differences in filtration efficiency among brands of masks.

Evaluation of four face masks after clinical use – The Aseptex mask,‡ Dental Surgical mask#, and Magic Arch#

masks were tested. Two general dentists and their primary chairside assistant wore coded face masks during patient treatment for one, two, three, and seven hours. Test periods were assigned randomly and repeated about eight times. No attempt was made to alter patient treatment. After each test, the mask was removed without contacting its surface, and BFE testing was performed. Mean BFE percentages were computed for each interval. One-way AOV and the Bonferroni multiple comparison procedure (alpha = 0.05) were performed to determine differences among intervals during which masks were worn, and between brands of masks tested and persons wearing the masks.

Performance of 6 face masks in humidity > 90 percent – ADDS-Air-Flow laboratory mask,** ADDS-Vanced laboratory mask**, Aseptex mask, Aseptex Plus mask, Duckbill surgical mask,‡ and the Dental surgical mask# were tested. Two subjects (a woman 54 kg and a man 100 kg) wore each of the test masks for 30 minutes in a chamber (42 inches x 24 inches x 24 inches). Their heads and shoulders were positioned inside the chamber, 6 and 12 inches, respectively, from an air-slurry polisher (Prophy Jet 30‡‡) operated on medium water and powder settings with an air pressure of 80 psi. After 30 minutes, the masks were removed without contacting their surfaces and BFE testing was performed as described above. BFE percentages were computed and plotted against mask type.

Dye tracer testing – The Aseptex mask, Magic Arch mask, Cover shield,## and Op-d-Op shield*** were tested. A test also was performed using the Aseptex mask in combination with the Op-d-op shield.

A subject's head and shoulders were positioned inside a chamber (30 inches x 30 inches x 36 inches) with a nebulizer (Micro Mist Aerosolizer #P220†††) emitting a dye (Blue Crayola Tempera‡‡‡) diluted with three parts Crayola Tempera‡‡‡ deionized water. Mean particle size of the airborne dye was 4.8 µm. The distance between the facial barrier and orifice of the nebulizer was 6 inches (Fig. 3).

Figures 4a to 4i illustrate the steps in the test procedures. A 2 inch x 2 inch cotton-filled gauze sponge (Healthco No. HCOC535051###) opened at its center was positioned so the nose was covered (Fig. 4g). Impermeable plastic was placed such that, for each facial barrier tested, inhaled air was allowed to enter:

(1) through the mask and peripheral border (Fig. 4c);

(2) through the mask material only (Fig. 4g); and

(3) through the peripheral border only (Fig. 4h). Figure 4i shows the results of a positive control test in which no facial barrier was worn. All tests were conducted for 10 minutes.

The gauze nose wraps from all but the combination Aseptex Mask/Op-d-op Shield tested were ranked according to area and intensity of dye stain by 21 evaluators who were not associated with the study. Kendall's coefficient of concordance was computed to determine agreement between evaluators.

Results

Bacterial filtration efficiency testing – In Figure 5, the 42 face masks tested are ranked in order of highest to lowest mean BFE scores. Lines connecting brand names indicate statistically non-significant scores, and overlapping lines indicate statistically similar groups. The test masks separated into two non-overlapping significance groups. The high filtration group included 22 face masks with mean BFE scores of 74 to 98 percent. All but two masks in this group were soft, pleated, rectangular masks. Two rigid, preformed, cup-style masks with a unique design (the ADDS-Air Flow laboratory and ADDS-Vanced laboratory masks) had filtration values of 91 to 92 percent. The scores of the 20 remaining masks were significantly lower (13 to 51 percent). In fact, all brands of conventional, cup-style face masks, including the Aseptex and Aseptex Plus brands, were in the low filtration group.

Figure 6 shows mean BFE percentages for 4 face masks worn during clinical treatment. The two soft, pleated, rectangular masks (Dental Surgical and Magic Arch) had efficiency scores of 97 to 99 percent, regardless of length of time worn. The Aseptex Plus proved to be statistically more efficient than the Aseptex. No statistical differences were observed with respect to persons wearing the mask.

Figure 7 shows the mean BFE percentages for 6 face masks tested under normal (about 50 percent humidity) and humid conditions (> 90 percent humidity); comparison of BFE percentages shows mask filtration was not

affected adversely by high humidity. The size and gender of the subject also had no effect on BFE scores.

Dye tracer testing – Figure 8 shows the gauze sponges worn over the test subject's nostrils during 10-minute dye tracer tests. Kendall's coefficient of concordance (0.993) showed high agreement between the 21 evaluators who ranked the sponges. In Figures 8a to 8i, the gauze sponges are shown as ranked, from least to most effective, by the evaluators. Results indicate the following:

(1) All test barriers blocked some airborne dye and proved more effective than no barrier (compare control gauze in Fig. 8a with all others).

(2) The face shields were least effective in blocking airborne dye (compare Fig. 8b and 8c with all others).

(3) The high-filtration mask provided the best defense against penetration (compare Fig. 8g, 8h, and 8i with all others).

(4) High-filtration material and close peripheral fit were needed for optimum blockage of aerosols (compare Fig. 8i with all others).

Figures 9a and 9b show leakage of dye tracer onto the interior surface of a high-filtration mask after 10 and 30 minutes of use. This problem was evident to varying degrees in all test masks. The colored dye enabled us to observe a problem not clinically apparent, because most dental aerosols (such as saliva and water) are colorless. Mask penetration is highly significant because it can bring airborne organisms into contact with the wearer's lips, nostrils, and skin.

In an additional test in which the Aseptex mask was used in combination with an Op-d-op Shield, aerosol penetration was diminished only slightly (Fig. 10). The combination did not provide the level of protection offered by the high-filtration face mask (Fig. 8g to 8i).

Discussion

Several theories concerning face masks were disproven in this series of tests. For example, filtration efficiency was not decreased by use in a humid environment for up to 30 minutes or by clinical use up to 7 continuous hours. Filtration efficiency was not affected by gender or size of the clinician. In addition, this study showed clearly that the test masks were not equally effective in preventing penetration of airborne debris. Shields proved to be substantially inferior to masks, owing to their lack of peripheral fit, which was shown to be as important as filtration efficiency in blocking airborne debris.

Size of airborne particles is important regarding the effectiveness of face masks. Investigators have demonstrated that dental procedures generate large quantities of aerosols 3 μm and smaller.[6] Such particles remain suspended at the end of treatment,[6,7] and can penetrate to the alveoli of the lower respiratory tract,[12] where their infectivity is greatly increased.[13] Therefore, dentists and staff should use facial barriers that block particles of this size, but our results showed only about half the test masks met this criterion. Conventional, preformed, cup-style face masks had low filtration. When choosing masks, dentists must consider the fact that 50 percent or more of deleterious airborne particles can filter through

this type of mask. After reviewing these data, two face mask manufacturers redesigned their preformed, cup-style face masks to achieve high BFE ratings (Triple Layer by 3M and Surgical Comfort by Healthco). These masks are now available.

It is clinically relevant to establish the length of time a face mask can be worn. This investigation showed neither high humidity nor use over time affected significantly the filtration of the masks tested. Dye tracer tests indicated the critical factor is soak-through of airborne debris. This process, called wicking, poses a threat when the wearer's lips and nostrils come in contact with moist areas, thus establishing direct mucous membrane contact with organism-laden material. Current mask design does not allow clinicians to determine when wicking has occurred, because interior surfaces of masks are not stained visibly when wet by colorless fluids. Given present mask technology, we agree with Craig et al., who suggested mask changes after 20 minutes in aerosol and 60 minutes in nonaerosol environments.[18] These recommendations were based on culturing of masks worn clinically. When worn longer than the recommended times, the masks became impregnated with microorganisms and, thus, were a source of, rather than a barrier to, potential pathogens.

When gathering information on filtration efficiency of face masks, dentists need to know that efficiency ratings can be generated by two tests. One is the BFE procedure performed here; the procedure is well suited to dental use of face masks. The other is the Greene and Vesley test,[19] which measures filtration from the inside of the mask outward through use of large particles. This test

is relevant to surgeons whose primary concern is deposition of droplets from the operator onto the patient. Unfortunately, Greene and Vesley test percentages often are used to promote masks to dental personnel because such ratings are substantially higher due to use of larger-sized test particles.

Filtration efficiency ratings of preformed cup-style masks increased slightly after three hours of clinical use. This phenomenon is common in BFE testing of cup-style masks, and is thought to be caused by impingement of droplets onto the mask. Some pores are blocked, which temporarily increases filtration efficiency. Droplets then coalesce, reducing filtration efficiency.

Confidentiality agreements between manufacturers and distributors often result in very nearly the same product being sold in different packaging. In this study, testing of some similar masks was performed because, in some cases, positive identification of manufacturers could not be obtained. Testing was justified on the premise that data was needed for specific brand names for which manufacturers could not be identified positively. However, test results indicate similarity of manufacturing technology, rather than the manufacturer, was the key factor in mask filtration efficiency. Test masks were separated by BFE ratings into two distinct groups, although masks from more than two manufacturers were evaluated. With only a few expectations, the 42 masks were divided into a soft, pleated, rectangular, high-filtration group; and a rigid, preformed, cup-style, low-filtration group.

Conclusions

Bacterial filtration efficiency ratings here indicate 22 of the 42 brands of test masks ensure efficient blockage of airborne debris. Protective barriers against small-particle aerosols should be selected from among this group (Fig. 5). Preformed, cup-style face mask with a conventional design, and face shields all provided significantly less blockage of airborne particles.

The main factor in deciding when to discard a face mask of nay kind was degree of wicking. Because penetration of fluids such as water and saliva cannot be observed on the mask's interior surface, the recommendations of Craig et al. (to change masks after 20 minutes in aerosol and 60 minutes in nonaerosol environments) should be followed.[18] They showed that masks worn for longer periods became a source of infectious material.[18]

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**Dependable Scientific Glass Co., Salt Lake City, UT 84115.*

†Andersen 2000 Inc., Peachtree City, GA 30269.

‡3M Co., Dental Products Division, St. Paul, MN 55144.

#Alpha Pro Tech, Inc., North Salt Lake City, UT 84054

***American Diversified Dental Systems, Anaheim, CA 92806.*

††Baxter, Deerfield, IL 60015.

‡‡Dentsply, York, PA 17405.

##GRD Products, Grand Junction, CO 81501.

****Op-d-Op Inc., Roseville, CA 95678.*

†††Micro Mist Corp., Hudson, OH 44236.

‡‡‡Binney and Smith Inc., Easton, PA 18044.

###Healthco Inc., Boston, MA 02116.

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Table 1. Alphabetical list by brand name of 42 face masks and 2 face shields

Brand name	Company	Description
(Face masks)		
1. 8500 Non-Toxic particle mask	Colwell	Rigid, preformed cup, 1 elastic strap
2. ADDS-Air Flow laboratory mask	American Diversified Dental Systems	Rigid, preformed cup, 2 elastic straps
3. ADDS-Vanced laboratory mask	American Diversified Dental Systems	Rigid, preformed cup, 2 elastic straps
4. Asept-All Com-Fit mask	Sultan Chemists	Rigid, preformed cup, 1 elastic strap
5. Aseptex mask	3M	Rigid, preformed cup, 1 elastic strap
6. Aseptex Plus mask	3M	Rigid, preformed cup, 1 elastic strap
7. Classic surgical mask	Baxter	Soft, pleated rectangle, 4 ties
8. Cone Classic surgical mask	Baxter	Rigid, preformed cup, 1 elastic strap
9. Contour medical mask	Island Poly	Rigid, preformed cup, 1 elastic strap
10. D-Sep mask	Barrier Protection	Soft, pleated rectangle, 1 elastic thread
11. Dental procedure mask	Alpha Dental mask	Soft, pleated rectangle, 1 elastic strap
12. Dental procedure mask	Healthco	Soft, pleated rectangle, 2 elastic ear loops
13. Dental surgical mask	Alpha Dental	Soft, pleated rectangle, 1 elastic strap
14. Disposable blue mask	Biotrol	Rigid, preformed cup, 1 elastic strap
15. Duckbill surgical mask	Baxter	Soft, duckbill rectangle, 4 ties
16. Dust & respirator mask	Biosafety Systems	Rigid, preformed cup, 1 elastic strap
17. Face mask frame with disposable insert	Biotrol	Rigid, preformed cup insert, 1 elastic strap over nose
18. Facelock wet mask	Alpha Dental	Soft, pleated rectangle, 1 elastic strap
19. Fluid guard surgical mask	Healthco	Soft, pleated rectangle, 2 elastic ear loops
20. Fluid shield surgical mask	Baxter	Soft, pleated rectangle, 4 ties
21. Fog-free surgical mask	Baxter	Soft, pleated rectangle, 4 ties
22. High-filtration isolation mask	Baxter	Soft, pleated rectangle, 2 elastic ear loops
23. Magic arch mask	Alpha Dental	Soft, pleated rectangle, 1 elastic strap
24. Molded mask	Kent Dental	Rigid, preformed cup, 1 elastic strap
25. Molded regular mask	Schein	Rigid, preformed cup, 1 elastic strap
26. Molded surgical mask	Professional Discount Mart	Rigid, preformed cup, 1 elastic strap
27. Pleated mask	Professional Discount Mart	Soft, pleated rectangle, 4 ties
28. Pleated form fit mask with ear loops	Kent Dental	Soft, pleated rectangle, 2 elastic ear loops
29. Polyfil air mask	Kent Dental	Plastic foam strip, 2.5 inch diameter holes serve as ear loops
30. Preformed cup mask with fluid guard	Cottrell	Rigid, preformed cup, 1 elastic strap
31. Protec-Shield mask	Alliance	Rigid, preformed cup, 1 elastic strap
32. Protec-Shield flat mask	Alliance	Soft, pleated rectangle, 2 elastic ear loops
33. Shield-pleated ear-loop mask	Cottrell	Soft, pleated rectangle, 2 elastic ear loops
34. Shieldmate mask	Cottrell	Soft, pleated rectangle with clear plastic eye covering attached, 4 ties
35. Sigma designer mask	Endicott & Beaman	Soft, pleated rectangle, 4 ties
36. Soft, ear-loop mask	Biosafety Systems	Soft, pleated rectangle, 2 elastic ear loops
37. Surgical antifog mask	Healthco	Soft, pleated rectangle, 2 elastic ear loops
38. Surgical mask	Healthco	Rigid, preformed cup, 1 elastic strap
39. Surgine II mask	Johnson & Johnson	Soft, pleated rectangle, 4 ties
40. Surgine II antifog mask	Johnson & Johnson	Soft, pleated rectangle, 4 ties
41. Tie-on surgical mask #1818	3M	Soft, pleated rectangle, 4 ties
42. Ulti-mask	Baxter	Soft, pleated rectangle, 4 ties
(Face shields)		
1. Cover shield	Glen R. Dean	Clear, rigid, plastic; molded to fit plastic glasses frame and facial contours
2. Op-d-op shield	Healthco	Clear, rigid, plastic attached to plastic head visor

REFERENCE NO. 6

VOLUME 19, ISSUE 9 – SEPTEMBER 1995
CRA Newsletter

SUBJECT: OPERATING GLOVES, IMPORTANT NEW TRENDS

Never before have so many *dental* clinicians used operating gloves routinely. Extensive use has revealed sensitivities not anticipated. *Glove manufacturers are now seeking alternatives because glove sensitivity can force clinicians to cease practice.* This report on new *powder free* alternatives focuses on: (1) Quality, clinical characteristics, & cost of 32 brands of *powder free* gloves; & (2) Clinical & lab data that strongly implicate glove defects in virus transfer.

METHODS:

A. GLOVE ACQUISITION – 6 boxes of about 100 gloves each from 32 powder free glove brands were acquired from 20 sources in 12 U.S. states from January through March '95.

B. CLINICAL CHARACTERISTICS TEST – 300 each of the 32 glove brands (9600 total) were used during routine clinical treatment by dentists & auxiliaries at 29 clinical sites in 19 U.S. states & rated according to: (1) Fit, (2) Tactile sensitivity, (3) Taste, (4) Tackiness when wet, (5) Resistance to tearing, & (6) Cuff length (adequate to pull *over* long sleeve).

C. MANUFACTURING DEFECTS TESTS – About 290 each of the 32 glove brands (9280 total) were tested *in-vitro* for perforations (FDA Water Leak Test) & 9 of each brand (288 total) were tested for time before hydration occurred (Dental-Alert Monitor by Novatec [713] 266-1976).

1. GLOVE QUALITY (defects & hydration time), CLINICAL CHARACTERISTICS, & COST OF 32 POWDER FREE GLOVES.

Gloves are ranked best to worst by *combination* of low glove defects & hydration times reaching 30 minutes.

GLOVE NAME, ORDER NUMBER & (SIZES)	GLOVE DEFECTS	HYDRATION TIME	CLINICAL RATING*	RETAIL COST /GLOVE/CASE
Biogel-D, 100-(51/2-9) (COTROL)	0.7%	28 min.	5.2	62¢ CONTROL
Crosstex Powderless, CTPLG-(S-M-L)	1.0%	29 min.	4.7	10¢
Ansell Conform, 114-(S-M-L)	2.4%	30 min.	4.6	11¢
Golden Glove, (no order #)-(S-M-L)	2.4%	30 min.	5.1	8¢
Aladan Tri-Clean 110, 0225240-(S-M-L)	3.8%	30 min.	4.2	12¢
Microflex Diamond Grip, G-4013-(S-M-L)	1.4%	22 min.	5.2	9¢
Tronex P.F., 3010-(XS-S-M-L-XL)	1.4%	22 min.	4.5	9¢
QualiTouch Powder Free, 40-95-(XS-S-M-L)	2.8%	26 min.	4.9	11¢
Royal Shield, (no order #)-(XS-S-M-L)	4.4%	30 min.	5.1	6¢
Latex Medical Exam Powder Free,100-(S-M-L)	4.6%	30 min.	4.0	10¢
Pharmaseal Flexam, 8878-(S-M-L)	4.6%	30 min.	4.4	7¢
Alpine Powder Free, (no order #)-(S-M-L)	1.4%	20 min.	4.6	9¢
Tillotson Powder Free Plus, (02-450-(S-M-L)	3.0%	25 min.	4.7	10¢
SS White Powder Free, 740-(XS-S-M-L)	2.8%	24 min.	4.6	11¢
MedPac Powder Free, 2000-(S-M-L)	5.1%	30 min.	4.9	10¢
Safeskin Satin Plus Powder Free, SP2-(XS-S-M-L-XL)	3.4%	25 min.	4.5	13¢
Safeskin Powder Free, 6301097-(XS-S-M-L)	2.7%	21 min.	4.6	12¢
Bio-Flex Gel Coat, BPF-(XS-S-M-L)	2.4%	19 min.	5.3	10¢
Life Guard Powder Free, 120-(S-M-L)	3.4%	20 min.	4.5	9¢
Sime Powder Free, 2000-(XS-S-M-L-XL)	0.7%	11 min.	4.5	6¢
Safetex P.F., 104-930-03-(S-M-L-XL)	2.1%	13 min.	4.8	11¢
Defend Latex Powder Free, 16-211-(XS-S-M-L)	5.1%	17 min.	4.5	11¢
Textured Powder Free, 18PFT-(S-M-L)	5.8%	17 min.	4.5	9¢
Dash Powder Free, (no order #)-(XS-S-M-L)	2.1%	2 min.	5.1	8¢
Arista Powder Free, PF208-(XS-S-M-L-XL)	4.2%	3 min.	4.5	8¢
Cranberry Powder Free, 16-201-(XS-S-M-L)	6.4%	9 min.	4.6	9¢
Exam Plus Powder Free, BS03-(XS-S-M-L-XL)	5.1%	2 min.	4.4	9¢
Skin-Gard P.F., 070-(XS,S-M-L)	10.8%	12 min.	4.9	7¢
Partners Powder Free, GWZS-(S-M-L)	10.8%	2 min.	4.5	6¢
PM Vinyl Powder Free, PMVPF-(S-M-L-XL)	— §	0 min. ¶	3.8	9¢
Durasafe Vinyl Powder Free, 223-(S-M-L-XL)	— §	0 min. ¶	3.3	9¢
Defend Vinyl Powder Free, 18-121-(S-M-L)	— §	0 min. ¶	3.9	12¢

§ FDA Water Leak Test does not discriminate defects in vinyl gloves.
¶ Vinyl is porous & allows immediate hydration
* On scale of 1-7, 7=BEST

BEST GLOVES OVERALL ARE Biogel-D, Crosstex Powderless, Ansell Conform, Golden Glove & Aladan Tri-Clean 110. LEAST EXPENSIVE, BEST OVERALL GLOVE is Golden Glove.

2. CLINICAL & LAB DATA THAT STRONGLY IMPLICATE GLOVE DEFECTS IN VIRUS TRANSFER.

PREMISE: Gloves are worn by clinicians to prevent exposure of patient & clinician to each other. *Integrity* of the barrier is first concern.

> *IN VIVO* LEAKS DEMONSTRATED: A Los Angeles surgeon who infected 18 of 142 patients (13%) in 10 months with hepatitis B theorized that finger irritation & pressure from suture tying might be implicated in virus transmission. Tests showed hepatitis B virus in washings from *inside* of gloves he wore while tying sutures, indicating he shed virus through skin on his fingertips. These viruses (42 nm), shed through irritated skin & escaping through defects in glove fingers provide first plausible explanation of disease transfer between patients & a clinician practicing Universal Precautions. (New York Times, March 22, 1994, C3)

> *IN VITRO* LEAKS DEMONSTRATED: Investigators testing 240 latex & 240 vinyl exam gloves found leakage of test virus ϕX174 in both types of gloves, regardless of whether gloves were just removed from box or stressed in 3 levels of simulated use. At heaviest use level, vinyl gloves leaked virus 9 times more frequently than latex gloves tested. (Korniewicz, D.M. et al., J Clin Micro 1990; 28:787-788)

****WITHOUT EFFICACIOUS INFECTION CONTROL PRODUCTS, THERE IS NO CONTROL OF INFECTIOUS DISEASES.**

CAUTIONS: A. Chlorination treatment is most common way to make powder free gloves. Process is *conceptually* good because it lowers protein antigens. In practice it is often done poorly to save time & money, & results in shortened shelf life, increased defects, slippery surface, foul taste & smell on gloves, brittle & cracking fingernails on clinician.

B. Vinyl is *porous*.

***CHOOSE POWDER FREE GLOVES CAREFULLY. STORE COOL, & USE WITHIN 3-4 MONTHS.*

3. CRA CONCLUSIONS:

Dental clinicians need to be aware that routine use of operating gloves can result in various hypersensitivity symptoms. Prevention is superior to searching for solutions after symptoms develop. New option is *powder free* gloves. Outstanding gloves in evaluation are listed above under BEST GLOVES.

REFERENCE NO. 7

VOLUME 16, ISSUE 10 – OCTOBER 1992
CRA Newsletter

SUBJECT: GLOVES, NON-STERILE OPERATING – UPDATE REVIEW

One year ago, CRA reported on quality & cost of 6,000 non-sterile operating gloves representing 20 brands used by dental clinicians (see Sep. '91 CRA Newsletter). Only 4 of 20 brands tested had *combination* of reasonable cost, overall good clinical characteristics, & low defects (7 of 20 brands met FDA's specification of 4.0% or less defects). Since both clinicians & patients depend on operating glove quality as primary infection control barrier, 1 year later the same tests were performed on all gloves still available to check for improvements. Following report is written in style similar to '91 report to facilitate comparisons.

1. TEST METHODS.

(a) GLOVE PURCHASE – Three boxes containing 100 gloves each of 24 brands of operating gloves were purchased from 36 distributors in 12 states in April, May, June '92. These included brands from the '91 evaluation, several new brands, & gloves from 1 manufacturer sold by 3 different distributors.

(b) CLINICAL CHARACTERISTICS TESTING – Seven characteristics evaluated were: (1) taste, (2) powder amount & texture, (3) tackiness after wetting, (4) tactile sensitivity, (5) resistance to tearing, (6) fit, & (7) cuff length & width.

(c) MANUFACTURING DEFECTS TESTING – FDA *in vitro* Water Tight Test was performed on 7,006 gloves to identify manufacturing defects. (All 7,200 gloves expected in this test were not available for testing due to presence of less than 100 gloves in 24 of the 72 boxes. One box contained only 54 of expected 100 gloves!)

2. **RESULTS.**

Refer to chart on page 2 for brand names that correspond to number abbreviations below & for details of evaluation.

(a) Gloves with **best *combination*** of reasonable cost, good clinical characteristics, & low manufacturing defects (pinholes) were defined as having cost of 8 cents or less, overall "good" clinical rating, & 4% or less defects. By these criteria #'s 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 13, & 17 were noteworthy.

(b) Gloves with **best *combination*** of above characteristics both in '92 & '91 were #'s 1 & 3.

(c) Gloves with **least manufacturing defects** (pinholes) were #'s 1, 4, 9, & 12.

(d) Gloves with **least cost (6 cents/glove)** were #'s 1, 2, 3, 5, 11, 15, & 18.

(e) Comparing '92 to '91 evaluation, following was observed:

(1) Significantly fewer manufacturing defects were observed (i.e. 2.5% or 176 total defective gloves out of 7,006 tested in '92 vs. 6.6% or 392 total defective gloves out of 5,937 tested in '91).

(2) Clinicians desiring gloves with 4.0% or less manufacturing defects (FDA maximum allowable) have more brands from which to choose in '92 (i.e. 19 in '92 vs. 7 in '91).

(3) Clinicians desiring gloves with overall **best *combination*** of reasonable cost, good clinical characteristics, & least manufacturing defects have more brands from which to choose in '92 (i.e. 12 in '92 vs. 4 in '91).

(4) No gloves were rated excellent in all clinical characteristics in '92 (i.e. 0 in '92 vs. 2 in '91).

3. **OBSERVATIONS:**

(a) More boxes of gloves lacked the expected 100 gloves than noted in previous tests (24 of 72 boxes). Most problematic were glove #'s 1, 10, 12, & 15.

(b) Cuffs on about half of gloves tested were too short to remain over fitted uniform sleeve to provide continuous barrier from hand onto arm.

(c) Some glove designs have recognized need for anatomically placed thumb on non-sterile gloves to relieve pressure on hand (glove #'s 17 & 21). Glove 23 was nitrile rubber rather than latex, which conforms to hand better & relieves pressure.

(d) Many glove brands now make "hypoallergenic" claim. CRA questions whether any glove made from latex can be truly hypoallergenic. In future, FDA may set

standards for gloves making this claim, but currently no standards exist.

(e) Since manufacturing defects continue to be a consideration, methods are needed for clinicians to test gloves before use &/or apply something to gloved hands to seal surface.

4. CRA CONCLUSIONS:

Overall quality of non-sterile operating gloves tested has improved substantially over last year. Twelve of 24 brands had combination of reasonable cost, good clinical characteristics, & lowest manufacturing defects – a 30% improvement over '91 test results. One glove (S.S. White Latex Dental Exam Gloves) had *no* manufacturing defects in 300 gloves tested, which demonstrates holes in gloves are not inevitable. CRA urges all glove manufacturers to strive for consistent zero defects & improve clinical characteristics.

5. GLOVES TESTED‡

GLOVE NAME & ORDER NUMBER‡	SUGGESTED RETAIL COST	CLINICAL RATING§	GLOVE DEFECTS	OVERALL RATING‡
*1. Bio-Flex Dental Examination Gloves #BIO	$5.95/100 Case of 20 boxes (6¢/glove)	Good	1.0%	3.0
2. Ammex Latex Exam Gloves #AM721	$5.95/100 Case of 10 boxes (6¢/glove)	Good	1.7%	3.2
*3. APO Health Non-Sterile Latex Examination Gloves #Natural Color	$6.00/100 Case of 20 boxes (6¢/glove)	Good	2.0%	3.3
4. Ammatex DispoMed Exam Gloves #LX-102	$7.50/100 Case of 10 boxes (7.5¢/glove)	Good	1.0%	3.5
5. Sime Disposable Examination Gloves #10001 10003	$5.95/100 Case of 20 boxes (6¢/glove)	Good	2.4%	3.5
6. "Audra Gloves You" Disposable Latex Gloves #1	$7.50/100 Case of 10 boxes (7.5¢/glove)	Good	1.3%	3.6
7. RubberCare Latex Examination Gloves #8050	$6.90/100 Case of 10 boxes (6.9¢/glove)	Good	2.0%	3.6
8. Glovetex Latex Examination Gloves #1012	$6.45/100 Case of 10 boxes (6.5¢/glove)	Good	2.4%	3.6
9. S.S. White Latex Dental Exam Gloves #95001	$8.00/100 Case of 10 boxes (8¢/glove)	Fair	0.0%	3.7
10. Aladan Latex Medical Gloves #1002	$7.50/100 Case of 10 boxes (7.5¢/glove)	Good	1.7%	3.7
11. Silky Touch Hypoallergenic Latex Examination Gloves #LG-ST-Med	$5.95/100 Case of 10 boxes (6¢/glove)	Good	3.1%	3.7
12. Healthco Disposable Latex Examination Gloves #HCOG101001 & HCOG101011	$7.50/100 Case of 20 boxes (7.5¢/glove)	Fair	1.0%	3.8
13. Pharmaseal Flexam Floor/Exam Latex Gloves #8857	$7.28/100 Case of 20 boxes (7.3¢/glove)	Good	2.4%	3.9

14. Ansell Disposable Latex Examination Gloves #HCOG100011 & HCOG100021	$6.50/100 Case of 20 boxes (6.5¢/glove)	Fair	2.4%	4.0
15. Chatsworth Latex Examination Gloves #8000 & 8001	$5.95/100 Case of 20 boxes (6¢/glove)	Good	4.2%	4.1
16. Safeskin Hypoallergenic Latex Examination Gloves #300 & 200	$7.45/100 Case of 10 boxes (7.5¢/glove)	Fair	2.0%	4.2
17. Naturalfit Latex Dental Exam Gloves No order #	$9.45/100 Case of 20 boxes (9.5¢/glove) (Canadian cost)	Good	1.7%	4.4
18. Columbia World Latex Examination Gloves #C102	$5.99/100 Case of 10 boxes (6¢/glove)	Fair	4.4%	4.5
19. Defend Hypo-Allergenic Latex Examination Gloves #LG-1003 & LG-1004	$6.95/100 Case of 20 boxes (7¢/glove)	Good	4.8%	4.6
20. Micro-Touch Latex Medical Gloves #5741 & 5721	$10.67/100 Case of 10 boxes (10.7¢/glove)	Fair	1.3%	5.0
21. QualiTouch Left/Right Latex Exam Gloves #40-865	$11.95/100 Case of 10 boxes (12¢/glove)	Good	3.1%	5.7
22. Protector Latex Examination Gloves #EX-2	$6.50/100 Case of 10 boxes (6.5¢/glove)	Good	8.7%	5.7
23. Nitril N-Dex Gloves #6005M	$15.00/100 Case of 20 boxes (15¢/glove)	Good	2.4%	6.5
24. Natraflex Ambi Latex Exam Gloves #8881-498706	$15.50/100 Case of 10 boxes (15.5¢/glove)	Fair	4.8%	7.8

‡ Overall rating is result of averaging across cost, 7 clinical characteristics, & mean manufacturing defects in 3 boxes of gloves. Low number = best grade.

* Indicates gloves from both '91 & '92 evaluations that had combination of reasonable cost, overall good clinical characteristics, & low manufacturing defects.

§ Each of 7 clinical characteristics listed on page 1, item 1 (b) were graded separately (Excellent=1, Good=2, Fair=3, Poor=4) & an overall mean determined.

CAUTION: Be careful if attempting to purchase gloves on above chart if purchasing from distributors other than those listed because gloves may not be same as those tested by CRA.

REFERENCE NO. 8

VOLUME 15, ISSUE 9 – SEPTEMBER 1991
CRA Newsletter

SUBJECT: GLOVES, NON-STERILE LATEX

Use of operating gloves by dental clinicians has become standard practice in U.S. AIDS epidemic & recent government regulations (OSHA) are factors responsible for shift from bare hands to gloves. Today, question is not "Should I wear gloves?" but "*WHICH GLOVES* should I wear?". Choices are based heavily on cost, but clinical characteristics also influence decision. However, most important factor – *absence of holes* – generally is not considered because clinicians have no way to detect small defects called pinholes, where molten latex failed to fuse during manufacturing process. U.S. government (FDA) has set 4% as maximum pinhole defects allowable in non-sterile latex gloves. Unfortunately, FDA tests that detect pinholes render gloves unsuitable for clinical use. To identify glove brands with least pinholes, most desirable clinical characteristics, & most reasonable cost, CRA purchased 6,000 gloves from all over U.S. & subjected them to testing. Below report includes: (1) test methods; (2) results & observations; (3) CRA recommendations on glove use; (4) gloves tested (page 2 chart); & (5) CRA conclusions.

1. TEST METHODS.

To consider 3 areas of interest to clinicians concerning gloves (cost, clinical characteristics, defects [pinholes]), CRA:

(a) Surveyed 10,000 dental clinicians to determine most popular gloves & most common sources for purchase (Dec. '90 CRA Newsletter).

(b) Purchased 6,000 gloves from 18 distributors located in 13 U.S. states.

(c) Tested clinical characteristics (taste, powder amount & texture, tackiness after wetting, tactile sensitivity, resistance to tearing, & fit) in controlled in-house & field tests.

(d) Performed *in vitro* test for manufacturing defects prescribed by FDA (Water Tight Test) on 20 different glove brands using 100 gloves from each of 3 different boxes. Every attempt was made to secure 3 different lot numbers. Lot distribution among brands was: 11 with 3 different lots, 2 with 2 different lots, & 6 with only 1 lot. Gloves were tested in multiple replicates (1 replicate = 1 glove from each of 60 boxes in random order).

2. RESULTS & OBSERVATIONS.

Chart on page 2 gives details of evaluation. Below is summary of results:

(a) Gloves with ***combination*** of low pinhole defects, most of desirable clinical characteristics, & reasonable cost were #'s 2, 3, 5, 6 on page 2 chart.

(b) In pinhole defects, 8 of 20 glove brands tested met FDA specifications of 4% or less. They are listed as #'s 1-8 on page 2 chart.

(c) In clinical characteristics, 3 of 20 glove brands were rated excellent (#'s 1, 2, 5 on page 2 chart), 12 were rated good (#'s 3, 6, 8, 9, 10, 11, 12, 15, 17, 18, 19, 20 on page 2 chart), & 5 were rated fair (#'s 4, 7, 13, 14, 16 on page 2 chart).

(d) Left/Right Latex Exam Gloves sold by Smart Practice had least defects, best quality across lots, & most novel design. This was only glove in study constructed on "clutched hand" mold rather than flat hand mold. Purpose of this innovation is to relieve continuous stress on hand muscles when gloved fingers are in working position on instruments.

(e) Nitrile N-Dex Gloves sold by Safeware Supplies was only glove in study made of nitrile rubber rather than latex. Although it did not pass FDA specifications for maximum pinhole defects in new gloves with 8.3% defects, tests showed it was considerably more resistant to puncture during use.

3. CRA RECOMMENDATIONS ON GLOVE USE.

(a) Wash hands well before putting gloves on & between glove uses to lower microbe counts. Warm, moist environment created by gloves accelerates microorganism activity on skin. Use antiseptic demonstrated rapid antimicrobial activity plus residual & cumulative action. CRA tests show 4% chlorhexidine gluconate formulations meet these criteria best. Some brand names include Bactoshield, CHG, Excelle, Hibiclens, Luroscrub, Novoclens, & Steri-Stat.

(b) Wear long sleeved uniform & purchase gloves with long enough cuff to allow gloving *over uniform sleeve*. Purpose of gloves & uniform is to provide barrier between environment & bare skin. Bare arms compromise this goal.

(c) Today, patients want assurance operator's gloves are used only in their treatment. Plan to glove & deglove in front of each patient.

(d) Operating gloves should not be used for operatory cleanup because design that allows tactile sensitivity is not as resistant to puncture as utility gloves.

4. GLOVES TESTED

* Order of best pinhole percent. ** Pg. 1, section 1, item (c) lists characteristics considered.

GLOVE NAME & ORDER NUMBER*	COST PER GLOVE FROM CRA SOURCE	CLINICAL RATING **	PERCENT DEFECTS IN 300 GLOVES
1. Left/Right Latex Exam Gloves #40870 (Clutched Hand Mold)	$11.95/100 12¢ each Case of 10 boxes of 100	Excellent	0.7
2. Non-Sterile Latex Examination Gloves #APOMW	$6.00/100 6¢ each Case of 20 boxes of 100	Excellent	1.3
3. Latex Exam Gloves now called Bio-Flex #B1000	$6.00/100 6¢ each Case of 20 boxes of 100	Good	2.0
4. Natraflex Ambl Latex Exam Gloves #8881-498706	$12.30/100 12¢ each Case of 10 boxes of 100	Fair	2.4
5. Micro-Touch Latex Medical Gloves or Dental Gloves #5721 or 5720-00	$8.50/100 9¢ each Case of 10 boxes of 100	Excellent	2.7
6. All Latex Multi-Purpose Ambidextrous Disposable Gloves #C-102	$5.99/100 6¢ each Case of 10 boxes of 100	Good	2.7
7. Defend Latex Examination Gloves #LG-1004	$8.95/100 9¢ each Case of 20 boxes of 100	Fair	3.7
8. Disposable Latex Examination Gloves #HCOG100021 & HCOG100011	$7.95/100 8¢ each Case of 20 boxes of 100	Good	4.3
9. Pharmaseal Flexam Floor/Exam Gloves #8857	$5.99/100 6¢ each Case of 10 boxes of 100	Good	5.4
10. Glovetex Disposable Ambidextrous Natural Latex Examination Gloves #3030	$6.45/100 7¢ each Case of 10 boxes of 100	Good	5.7
11. Disposable Latex Examination Gloves— Ambidextrous Unisize #HCOG101001	$7.95/100 8¢ each Case of 20 boxes of 100	Good	6.0
12. Latex Medical Gloves #1002	$7.95/100 8¢ each Case of 10 boxes of 100	Good	6.7
13. Disposable Examination Gloves #SL02	$5.95/100 6¢ each Case of 20 boxes of 100	Fair	7.0
14. Latex Examination Gloves No order #	$5.95/100 6¢ each Case of 10 boxes of 100	Fair	7.7
15. Nitrile N-Dex Gloves #7005	$9.80/100 10¢ each Case of 20 boxes of 100	Good	8.3
16. Latex Examination Gloves #M-1002	$5.00/100 5¢ each Case of 20 boxes of 100	Fair	9.3
17. Latex Examination Gloves #R1001	$8.95/100 9¢ each Case of 20 boxes of 100	Good	10.3
18. Disposable Non-Sterile Latex Examination Gloves #XGM	$5.90/100 6¢ each Case of 10 boxes of 100	Good	13.1
19. Disposable Latex Gloves No Order #	$7.50/100 8¢ each Case of 10 boxes of 100	Good	15.2
20. Latex Examination Gloves #8001	$5.90/100 6¢ each Case of 20 boxes of 100	Good	17.4

*CAUTION: Be careful if attempting to purchase gloves on above chart if purchasing from distributors other than those listed because gloves may not be same as those tested by CRA. Glove industry has complicated interwoven network that makes glove manufacturing source & grade impossible for clinicians to trace. To link responsibility for glove quality to manufacturing source, name & address of manufacturer should be listed on each box, but currently this is not required.

5. CRA CONCLUSIONS:

Gloves with *combination* of consistently low pinhole defects, most of desirable clinical characteristics, & reasonable cost are listed in chart above under following numbers: 2, 3, 5, 6. Most gloves tested (12 of 20 brands) exceeded 4% defects specified by FDA, indicating need for clinical method to test for defects *before* using gloves or material to spread over gloved hands to seal latex surface before use. Currently double gloving is only way to manage glove defect problem.

REFERENCE NO. 9

VOLUME 13, ISSUE 1 – JANUARY 1989
CRA Newsletter

SUBJECT: OPERATING GLOVES, UPDATE

STATUS OF DENTAL GLOVES IN U.S. Operating gloves are now used routinely by many dental clinicians. Primary factor in change from bare to gloved hands was AIDS epidemic which stimulated increased awareness of infectious diseases in general. Unexpected demand for gloves created shortages, increased prices, & decreased quality. However, currently supply is surpassing demand, & cost & quality are improving. In future, government regulations will stipulate criteria for both sterile & non-sterile glove quality.

GLOVES SELECTED FOR CRA EVALUATION. Glove origin must be identified by consumer to determine glove differences. This is difficult because a few manufacturers supply a large number of distributors. In order to conceal fact that same glove is sold under many different brand names for many different prices, name of glove manufacturer is generally kept confidential & each distributor uses unique brand name &/or label designs. This practice has serious implications for clinicians having hypersensitivity & fit problems. It also prevents check to ascertain if glove cost is commensurate with quality. To overcome these communication obstacles, chart on page 2 lists gloves tested alphabetically by *manufacturer's name.*

GOALS OF EVALUATION. CRA goal for this evaluation was to test gloves sold by well known distributors that represented all 6 major U.S. manufactures (Aladan, Ansell, Baxter/Travenol, Perry, Surgikos, Tillotsen), plus

manufacturers outside U.S. To locate your particular gloves in chart on page 2, call distributor where you purchased the gloves & obtain *manufacturer's name.* Refusal to disclose name of manufacturer generally indicates problems with quality or pricing.

CHARACTERISTICS TESTED & PROCEDURES USED. Glove characteristics tested were: (1) Manufacturing defects using 2 different methods (TEST METHOD 1 – ASTM Airtight Test, ANSI/ASTM D 3577-78a. Glove inflated with air [1.5kPa or ~ 0.657 psi] & submerged in H_2O so longest finger is about 200 mm from surface & tester checks for escaping air. TEST METHOD 2 – Ballbach Air Inflation Test, Ballbach R.L., et. al., J AOAC, 55(5)1074-1080, 1972. Uses protocol similar to ASTM Airtight Test except glove is compressed to force air into one finger at a time which places glove material under tension while tester checks for escaping air); (2) Smell; (3) Taste; (4) Amount of powder; (5) Tactile sense; & (6) Resistance to tear. Test results are reported in chart on page 2.

CRA OVERALL CONCLUSIONS:

Gloves with comparatively good quality were identified in all 5 categories tested. This information provides clinicians with tenable basis for glove selection. Clinical characteristics such as smell, taste, amount of powder, tactile sense, & tear resistance need improvement in most gloves tested. Chart on page 2 links names of manufacturers & distributors to specific gloves. This assists clinicians with hypersensitivity & fit problems to identify gloves with actual differences. Two brands of vinyl gloves tested demonstrated quality that enables individuals hypersensitive to latex gloves to consider

them as viable alternative. One copolymer over-glove had very low number of defects, making it possible to consider over-glove as way to avoid contamination when touching telephone, patient chart, x-rays, pencils, or pens, etc., during patient treatment.

EVALUATION OF OPERATING GLOVES

A	B	C	D		E				
MANUFACTURER	COUNTRY CONTROLLING PRODUCTION	COST PER GLOVE FROM REPRESENTATIVE U.S. DISTRIBUTOR	ASTM PERCENT DEFECTS *	BALLBACH PERCENT DEFECTS *	SMELL * *	TASTE * *	AMOUNT OF POWDER * *	TACTILE SENSE * *	RESISTANCE TO TEAR * *
LATEX, NON-STERILE									
1. Aladan	U.S.	16¢	0.7	15.1	F	G	E	G	P
2. Ansell	U.S.	13¢	1.6	9.4	G	F	G	G	F
3. Bantex Corp.	Taiwan	13¢	16.0	38.0	G	G	G	F	G
4. Baxter/Travenol	U.S.	15¢	2.3	8.0	G	G	G	E	E
5. Baxter/Travenol	U.S.	18¢	2.1	9.6	G	G	G	G	E
6. London Rubber Co	England	48¢	2.3	7.3	G	P	E	F	G
7. Perry	U.S.	20¢	4.0	9.0	G	P	G	P	G
8. Surgikos	U.S.	30¢	2.0	6.5	F	G	F	G	G
9. Surgikos	U.S.	19¢	3.0	8.3	G	F	F	G	G
10. Tillotsen	U.S.	13¢	1.0	16.7	F	F	G	G	P
11. Unidentified	Taiwan	—	41.3	65.2	E	G	F	G	P
12. Unidentified	China	—	8.0	28.0	G	G	G	G	P
13. Unidentified	Malaysia	13¢	3.3	15.3	G	G	G	G	P
LATEX, STERILE									
1. Baxter/Travenol	U.S.	35-40¢	1.5	5.5	G	G	G	G	G
HYPOALLERGENIC, STERILE									
1. Baxter/Travenol	U.S.	63¢	5.1	17.5	P	G	G	G	F
2. Baxter/Travenol	U.S.	$1.10	2.7	9.2	G	G	G	G	G
VINYL, NON-STERILE									
1. Baxter/Travenol	U.S.	10¢	NT	1.0†	G	G	E	E	G
2. Becton-Dickinson	U.S.	10¢	NT	2.0†	F	G	E	F	E
3. Becton-Dickinson	U.S.	11¢	NT	6.0†	F	F	E	G	G
COPOLYMER, NON-STERILE									
1. Armin Poly-Version	U.S.	5¢	NT	4.0†	E	E	E	E	—
2. Becton-Dickinson	U.S.	7¢	NT	9.0†	E	E	E	E	—
3. Unidentified	China	5¢	NT	11.1†	E	E	E	E	—
4. Unidentified	Taiwan	4¢	NT	25.3†	E	E	E	E	—

* Lots tested available upon request.

NT Not tested using ASTM Airtight Test because lack of elasticity precluded mounting procedures used in this method.

* * E= excellent; G= good; F=fair; P= poor

† Agreement has not been reached on standard test for these gloves. Defect percentages using Ballbach Air Inflation Test are deceptively low because glove material does not have same elasticity as latex & air compressed into fingers is under lower pressure.

TEST RESULTS BY GLOVE TYPE:

1. LATEX GLOVES – Most gloves tested met, or came close to meeting, ≤2.5% manufacturing defects using ASTM Airtight Test. However, if latex was stressed (Ballbach Air Inflation Test), percent manufacturing defects on all gloves tested increased *significantly*, indicating presence of weak spots &/or smaller holes not detected by ASTM procedure. Generally, best gloves had low ASTM & Ballbach percentages, & Ballbach percentages were only 3-4 times higher than ASTM percentages (ie: gloves listed alphabetically on chart above as Ansell, Baxter, London Rubber, & Surgikos). Some imported gloves came close to this criteria (Malaysia), but gloves tested from Taiwan & China had substantially higher number of defects. Clinical characteristics of all gloves tested need improvement.

2. VINYL GLOVES – Brands of vinyl gloves tested had significantly fewer defects than those tested by CRA in past using same method. However, test method used could be questioned because standard test for vinyl gloves is lacking. Baxter/Travenol & Becton-Dickinson Tru-Touch gloves could be alternatives for clinicians hypersensitive to latex gloves.

3. COPOLYMER OVER-GLOVES – Quality of Armin Poly-Version copolymer gloves was significantly better than expected for this type manufacturing process. These gloves could be useful worn over operating gloves during non-treatment tasks required during patient treatment such as handling x-rays, patient chart, telephone, etc. Gloved hands should be washed well before touching over-gloves to minimize cross-contamination.

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Leakage of Virus through Used Vinyl and Latex Examination Gloves

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A total of 480 examination gloves (240 vinyl and 240 latex) were stressed by using manipulations designed to mimic patient care. At the highest use level, 38 (63%) of 60 vinyl gloves leaked bacteriophage ϕX174 compared with 4 (7%) of 60 latex gloves. At lower use levels, there was no statistically significant difference in leakage.

Recently, there has been concern among health care personnel about the use of vinyl and latex gloves as barriers against the transmission of microorganisms. A recent outbreak of herpes whitlow among intensive care nurses who wore gloves[7] has focused attention on the integrity of gloves during use. We previously investigated vinyl and latex examination gloves as barriers against bacteria and found that both types of gloves provided some protection, but latex gloves maintained

integrity longer under in-use conditions.[6] Dalgleish and Malkovsky[2] have reported that the quality of latex gloves varies with the manufacturer and that latex gloves may not give total protection from human immunodeficiency virus penetration. In fact, gloves scanned by electron microscopy have shown 30- to 50- μm holes, suggesting that viruses could penetrate the gloves.[1] Degroot-Kosolcharoen and Jones[3] investigated the permeability to water and blood of sterile latex gloves and examination latex and vinyl gloves and found greater leakage in examination gloves. Health care personnel continue to question the barrier effectiveness of latex and vinyl examination gloves used in clinical practice. This study was conducted to determine whether bacteriophage ϕX174 could penetrate used vinyl and latex examination gloves after standardized manipulations.

Procedure for glove manipulation. Because of findings in our previous study (6), standardized manipulations designed to mimic patient care activities were performed on groups of gloves in advance of testing (Table 1). Participants wore no rings or nail polish and had their fingernails filed short and smooth. Sixty gloves of each type were tested at each use level. As positive controls, vinyl and latex gloves had either two or five holes punctured in the index finger with a 21-gauge hypodermic needle and were assayed for bacteriophage leakage. In addition, one latex glove was torn approximately 1 cm at the index finger.

Preparation and assay of phage. Stock suspensions of ϕX174 bacteriophage were prepared by growth in liquid culture with *Escherichia coli* C (ATCC 13706) at 37°C (5). The titer of ϕX174 was determined by plaque formation

using the tryptone top-agar layer method (5, 8). Phosphate-buffered saline (100 ml; pH 7.4) containing10^6 to 107 PFU of ϕX174 was poured into each glove. The quantity of liquid was enough to fill the glove to just about the fingers. Each glove was held over an empty collection container for 1 min and observed for leakage of liquid. Each glove was then draped for 4 min over the edge of a collection container such that the fingers were suspended in the interior of the container and the empty remainder of the glove fell over the exterior. Phosphate-buffered saline (100 ml) was poured into the collection container, and the fingers were immersed for 5 min more. Samples (1ml) of the collection buffer were obtained after up-and-down agitation and removal of the glove. In this study, a visible leak was defined as a leak observed by the naked eye. A viral leak was one in which virus was detected in the collection container. The effective volume leaked from each glove was calculated in microliters as the total number of virus particles in the collection container divided by the virus concentration inside the glove. The chi-square and the Fisher exact tests were used to assess differences in the proportions of gloves which leaked. The Mann-Whitney U test was used to test for differences in the volume of fluid leaked.

A total of 480 (240 vinyl and 240 latex) examination gloves were tested, with 60 each in the four categories of use. None of the latex gloves with needlestick holes had visible leaks or were positive for viral leakage. However, the torn latex glove was positive for both visible and viral leaks (5.0×10^6 PFU). The punctured vinyl gloves had both visible and viral leakage (two holes, 7.6×10^3 PFU; five holes, 4.9×10^6 PFU). All test gloves with visible

leaks also leaked virus (Fig.1). Virus leakage was found in both types of gloves: 55 (22.9%) of 240 vinyl and 18 (7.5%) of 240 latex gloves ($x^2 = 20.94$. $P = 0.00005$). In addition, in 34 (14.1%) of 240 vinyl gloves and 16 (7%) of 240 latex gloves, visible leaks were not observed, but virus still leaked through: 5×10^2 to 1×10^6 PFU per glove for vinyl and 5×10^2 to 1.3×10^4 PFU for latex.

There were not statistically significant differences in leakage rates for latex or vinyl gloves at use level 0, 1, or 2. However, as with visible leaks, there was a statistically significant difference in viral leaks at use level 3. Of the vinyl gloves, 63% (38 of 60) in use level 3 leaked virus compared with 7% (4 of 60) of the latex gloves (Fisher exact test, two tailed; $P = 0.000001$). There was a wide range in the volume leaked at each of the four use levels (Table 2). Vinyl gloves leaked more than latex gloves (Mann-Whitney U test: $P = 0.0001$).

This study demonstrated that some vinyl and some latex examination gloves permit viral leakage. At use levels 0, 1, and 2, the proportions with viral leakage were not significantly different between glove types. At use level 3, more vinyl gloves than latex gloves leaked virus, and the volume leaked was higher. The needlestick punctures in the fingertips of gloves which were meant to serve as positive controls resulted in visual and viral leaks in the vinyl gloves but not in the latex gloves. This result was unexpected and may have been caused by the elastic resealing property of latex.

The high proportion of gloves that leaked some amount of ϕX174 indicates that the effective hole size is greater than 27 nm, since that is the diameter of the virus

particle. These findings are consistent with the work of Kotilainen et al. who were able to detect leakage of poliovirus (20 to 30 nm in diameter) through a glove barrier. Further evidence suggests that microorganisms can pass from the outside of a glove, through the glove, and onto the hand.[4,6]

These results, from the testing of a single brand of vinyl gloves, suggest that holes can be formed during procedures related to routine patient care. Under the conditions of this study, latex gloves appeared to be less susceptible to hole formulation than vinyl gloves. Although these experiments should be expanded to include other brands of both types of gloves, our results suggest the need to change gloves after moderate periods of use. In addition, since there may be viral leakage during glove use, a conscientious handwashing technique should be used after glove removal.

Table 1. Procedures for preparation of gloves prior to test

Use Level[a]	Procedure
0	Remove from box
1	Don gloves and remove them
2	Levels 0 and 1 plus rub each gloved hand with a washcloth in the following sequence: palm each finger in a twisting motion, thumb, and back of hand
3	Levels 0-2 plus (I) attach a capped needle to a Luer-Lok syringe and then remove it 30 times; (ii) connect and disconnect

Luer-Lok syringe to intravenous tubing and manipulate a stopcock eight times; (iii) wrap, tape, and unwrap a blunt object two times to simulate bandaging an amputation stump

[a]Use level in previous paper refers to manipulation level (6).

Table 2. Volume leaked from vinyl and latex gloves

	Median leakage (μl: range)	
Use level	**Vinyl**	**Latex**
0	4.19 (0.34-8.03)	0.08 (0.02-227)
1	1.09 (0.08-1,250)	0.26 (0.02-0.54)
2	6.12 (0.11-1,250)	0.57 (0.03-840)
3[a]	229 (0.02-7,142)	2.20 (0.06-437)

[a]Mann-Whitney U-test differences in volume leaked between vinyl and latex gloves, $P = 0.000001$

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Survival of Hepatitis A Virus on Human Hands and Its Transfer on Contact with Animate and Inanimate Surfaces

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The survival of hepatitis A virus (HAV; strain HM175) on the hands of five volunteers was determined by depositing 10μl of fecally suspended virus on each fingerpad and eluting the inoculum after 0, 20, 60, 120, 180, and 240 min. The amount of virus recovered from each fingerpad at 0 min was approximately 6.0 x 10^4 PFU. At the end of 4 h, 16 to 30% of the initially recoverable virus remained detectable on the fingerpads. HAV inocula (10 μl; approximately 1.0 x 10^4 PFU) placed on fingerpads or 1-cm-diameter metal disks were used to determine virus transfer to clean surfaces upon a 10-s contact at a pressure of nearly 0.2 kg/cm^2. When the inoculum was dried for 20 min, virus transfer from fingerpad to fingerpad, fingerpad to disk, and disk to fingerpad ranged from 2,667 to 3,484 PFU, while 0 to 50 PFU could be transferred after 4 h of drying. Elevation of the contact pressure alone from 0.2 to 1.0 kg/cm^2

resulted in an approximately threefold increase in the amount of virus transferred. Incorporation of friction (10 half turns of the finger during 10 s of contact) with the low and high levels of pressure gave two- and threefold increased in the PFU of virus transferred, respectively. Pressure and friction were found to significantly affect HAV transfer ($F = 33.98$; $P < 0.05$), irrespective of the mode of transfer used. No statistically significant interaction was observed between mode of transfer and pressure or friction. The findings of this quantitative study suggest that human hands may play an important role in the direct as well as the indirect spread of HAV.

Infections caused by hepatitis A virus (HAV) are endemic throughout the world (6, 21, 41). Apart from community-based epidemics (7, 15, 16, 26), outbreaks of the disease are frequently associated with hospitals (3, 13, 25, 45), day-care centers (12, 15-17, 40), schools and institutions for mentally ill people (27, 31, 37, 41), and eating establishments (19, 25, 28, 30). Whereas fecally contaminated food (e.g., shellfish) and potable water are well recognized as vehicles for HAV (6, 8, 36, 41), in nearly 50% of the cases the vehicle(s) responsible for virus spread remains unidentified (15). Strong circumstantial evidence indicates that virus-contaminated hands play a major role in the spread of the virus, particularly in institutional settings (7, 19, 25, 26, 28, 30, 35).

For hands to be important HAV vehicles, the virus must remain viable on human skin. Previously, we have shown that HAV can survive on hard surfaces for prolonged

periods (33) and also that it is relatively resistant to many of the commonly available hard-surface disinfectants (32). Infectious virus transfer from contaminated to clean surfaces through contact has been reported (2, 14, 18, 20). Therefore, this study was designed to determine HAV survival on human hands and to assess how routine interactions of hands with other contaminated surfaces could further promote the spread of HAV. The study design also included determination of the role that inoculum age, pressure, and friction may play in the transfer of infectious HAV from contaminated to clean surfaces.

MATERIALS AND METHODS

Cells. A seed culture of FRhK-4 cells was kindly provided to us by M.D. Sobsey, University of North Carolina, Chapel Hill. The methods for the cultivation and maintenance of these cells have been described previously (32, 33, 47). Briefly, Eagle minimum essential medium (GIBCO, Grand Island, N.Y.) with 10% fetal bovine serum (GIBCO), 2 mM glutamine (GIBCO), 0.1 mM nonessential amino acids (GIBCO), 50μg of gentamicin sulfate (Cidomycin; Roussel, Montral, Quebec, Canada) per ml, 100 μg of kanamycin (GIBCO) per ml, 0.015 M HEPES (*N*2-hydroxyethylpiperazine-N′2-ethanesulfonic acid; GIBCO), and 0.113% sodium bicarbonate (BDH Chemicals, Toronto, Ontario, Canada) was used for growing the cells. The cells were maintained in the same medium, but the maintenance medium contained only 2% fetal bovine serum.

Virus. The HM-175 strain of HAV was also received from M.D. Sobsey. Stock virus was prepared by infecting

FRhK-4 monolayers at a multiplicity of infection of 0.01. The virus was allowed to absorb for 90 min at 37°C before the addition of maintenance medium and a further incubation at 37°C until 75 to 80% of each virus-infected monolayer was affected by virus cytopathology (4 to 5 days of incubation). The cultures were frozen (-20°C) and thawed three times, and the culture fluid was centrifuged for 10 min at 1,000 x g. The virus was concentrated 10-fold by polyethylene glycol hydroextraction as described by Ramia and Sattar (42). Briefly, dialysis tubing (Spectrum Medical Industries Inc., Los Angeles, Calif.) containing 100 ml of the clarified cell culture fluid was placed in a plastic tray and completely covered with polyethylene glycol (molecular weight, 8,000; Matheson, Coleman and Bell, Norwood, Ohio). After overnight hydroextraction at 4°C, the residue in the dialysis tube was resuspended in 2 ml of Earle balanced salt solution. The concentrate was divided into aliquots for storage at -70°C.

HAV plaque assay. Determinations of HAV PFU were carried out in FRhK-4 monolayers in 12-well plastic plates (Costar, Cambridge, Mass.) as described previously (32,33). Briefly, three wells were used for each virus dilution tested, and each well received 0.1 ml of the inoculum. HAV was then allowed to absorb for 90 min at 37°C. The overlay consisted of Eagle minimum essential medium, 2% fetal bovine serum, 2mM glutamine, 0.1 mM nonessential amino acids, 50µg of gentamicin sulfate per ml, 100 µg of kanamycin sulfate per ml, 2 µg of amphotericin B (Fungizone; GIBCO) per ml, 0.015 MHEPES, 0.113% sodium bicarbonate, 0.75% agarose (type II; Sigma Chemical Co., St. Louis, Mo.), and 26 mM

magnesium chloride (BDH). The plates were sealed in laminated plastic bags (Philips, Toronto, Ontario, Canada) and incubated for 8 days at 37°C. The procedure for fixing and staining the monolayers prior to counting plaques has been described previously (29).

Virus-suspending medium. The fecal samples used in this study was obtained from a healthy 5-month-old baby. A 10% (wt/vol) suspension of it was prepared in normal saline; it was clarified of gross particulate matter by centrifugation for 10 min at 1,000 x g and passed through a 0.2-μm-pore-size membrane filter (Nalge Co., Rochester, NY) to remove bacteria and fungi. The filtrate was found to be free of cytotoxicity and endogenous viruses when it was tested in FRhK-4 cells by the procedure described before (29). When HAV was diluted 1:10 in this fecal suspension and held at room temperature for 4 h, no loss in virus infectivity was observed.

Disks. Stainless-steel disks (diameter, 1 cm) were punched out of locally purchased no. 4 finish polished sheets (thickness, 0.75 mm) and were used to represent environmental hard surfaces. The procedure for their decontamination, cleaning, and sterilization before reuse has been described in detail before (44).

Volunteers. Permission to place the virus on the hands of adult volunteers was first obtained from our university's Ethics Committee. Any individual with cuts or abrasions on the hands was automatically excluded from the study. Each volunteer was then thoroughly briefed on the experimental protocol and the risks involved before being asked to sign a consent form. The age of the participants ranged from 26 to 45 years.

At the end of the experiment, the volunteer was asked to gently press the experimentally contaminated fingerpads on a piece of paper towel soaked in a 6% solution of sodium hypochlorite (Javex Canada Ind., Toronto, Ontario, Canada) for 3 min for their decontamination. The hands were then washed thoroughly with ordinary soap and running tap water and dried with a paper towel.

HAV survival on human hands. Virus survival on the hands of volunteers was tested by the procedure of Ansari et al. (2). The hands were first washed in lukewarm running tap water for 30 s, disinfected by thoroughly rubbing them with 70% ethanol (BDH), and then air dried. To demarcate the areas for virus deposition, each fingerpad was pressed hard over the mouth (8-mm inside diameter) of a empty plastic vial (no. 72.694.006; Sarstedt Inc., St. Laurent, Quebec, Canada).

Before each experiment, the stock virus was diluted 10-fold in the fecal suspension. Ten microliters of the fecally suspended virus was immediately placed, using a positive-displacement pipette (Gilson Medical Instruments, Villiers-le-Bel, France), into each of two glass vials containing 990 μl of Earle balanced salt solution; these acted as the input virus controls.

Ten microliters of the virus suspension was also deposited at the center of the demarcated area on each fingerpad. In each experiment, the amount of virus in the inoculum on the fingerpads (0-min control) was determined by immediately recovering the deposited virus separately from four fingerpads of the left hand, as follows. The contaminated area was placed over the mouth

of a vial identical to that used for fingerpad demarcation but containing 990 µl of Earl balanced salt solution. The vial was inverted, with fingerpad still over it, and held in position for 5 s. This was followed by 20 full inversions of the vial with the vial still in place and then an additional 5 s of soaking and 20 more full inversions. The surface of the fingerpad was then scraped on the inside rim of the vial to recover as much of the fluid as possible. This technique was found to recover approximately 6.0×10^4 PFU or 70% of the input infectious virus. To determine HAV survival on hands, virus was randomly eluted from separate fingerpads at 20, 60, 120, 180, and 240 min after their inoculation.

Five volunteer participated in the experiments on virus survival on hands, and each experiment was performed three times on each volunteer.

Contamination of disks and virus elution. Each clean and sterile disk received 10µl of fecally suspended HAV. In every experiment, the amount of infectious virus deposited on the disks (0-min control) was determined by placing two inoculated disks immediately into two separate glass vials containing 990 µl of Earle balanced salt solution. The vials were sonicated to elute the virus from the disks. This procedure recovered approximately 6.23×10^4 PFU, which represented nearly all of the infectious virus placed on each disk (33).

Pressure and friction. The effect of a low (approximately 0.2 kg/cm^2) and a high (approximately 1.0 kg/cm^2) level of pressure was tested in the virus transfer experiments. The low level of pressure, with or without friction, was taken to represent ordinary touching of

environmental surfaces. Animate-animate interaction through handshaking was represented by a pressure of 1.0kg/cm^2 without friction, while the same pressure with friction simulates opening of doors and other, equivalent actions.

Depending on the type of experiment, a clean or HAV-contaminated disk was placed at the center of the pan of a scale with a digital readout (model no. 1206 MP; Sartorius, Gottingen, Germany), and a virus donor or recipient fingerpad was pressed on it until the desired pressure was achieved and was held there for 10 s; pressure was computed from the balance reading and the area of the contaminated surface. For virus transfer from fingerpad to fingerpad, the recipient fingerpad was placed on the scale and placed into contact with the donor.

In an attempt to standardize the friction to be applied during virus transfer, each volunteer was first thoroughly briefed on and given the opportunity to properly practice the following procedure. Once contact was made between the disk and the fingerpad and the desired pressure level was attained, the finger was rotated in half circles 10 times over the 10-s period of contact; in the case of fingerpad-to-fingerpad transfer, the donor was required to apply friction.

HAV transfer experiments. The extent of infectious HAV transfer upon fingerpad-fingerpad interaction and fingerpad-environmental surface contact was examined as follows. After letting the virus inoculum dry on the donor surface of the desired length of time, contact was made at the required pressure, with or without friction, with the recipient surface for 10 s. Virus was then eluted

form the recipient as well as the donor surfaces. The two values thus obtained were added together to represent the total amount of infectious virus detectable on the donor before transfer, and the amount of virus transferred was determined as a percentage of this total. The disk-to-fingerpad and the fingerpad-to-disk experiments were conducted on each of two volunteers, whereas the fingerpad-to-fingerpad mode involved volunteer E as the donor and volunteer A as the recipient.

Statistical analyses. HAV survival on the fingerpad of each volunteer at each time point (x value) was expressed as a fraction of HAV eluted at 0 min. These observations were then normalized to the 0-min value as 100%. Such data were used to generate log-linear plots ($\log_{10}$) in a computer program (SigmaPlot version 4.0; Jandel Corp., Corte Madeira, Calif.). First-order linear regression lines were then fitted to the plots in the same program. HAV decay rates on the fingerpad were obtained from the gradient of the fitted regression lines as loss of virus infectivity titer in $\log_{10}$ PFU per minute. The half-lives were calculated from the decay rate constants (K_i), as discussed by Segel (46).

To analyze the data obtained from HAV survival and transfer between the three models (fingerpad-fingerpad, fingerpad-disk, disk-fingerpad) over 4 h by using two volunteers, the data were normalized as described above. Bar chart plots were then produced in the SigmaPlot computer program.

In order to assess the effects of pressure, friction, and the transfer mode used on the amount of HAV transferred,

the raw data were entered into an SAS program (Statistical Analysis Systems, University of North Carolina, Chapel Hill) and a three-way analysis of variance was performed. The possible interactions between the variables (pressure, friction, and type of model) were also analyzed. The Tukey test was performed post hoc in the same program. To compensate for differences in virus transfer because of time, an average of the values obtained with the 20- and 60-min HAV transfers was computed and used in the three-way analysis of variance.

RESULTS

HAV survival on human hands. The mean virus titer at 0 min on fingerpads in the HAV survival experiments was approximately 6.0×10^4 (range, 5.3×10^4 to 7.2×10^4) PFU in the 10-μl inoculum. The results of these experiments are presented in Fig. 1. The pattern of HAV decay on fingerpads appeared to be biphasic. Nearly 68% of the input infectious virus became undetectable within the first 60 min after contamination of the fingerpads. Even though this early loss in virus infectivity was particularly pronounced in volunteer E, the rate of decay was essentially similar during the 60- to 240-min period for all five volunteers. The amount of HAV detectable at the end of 4 h ranged from 16 to 30% of the initially detectable amount; the actual PFU recovered at the end of 4 h were 15,740 and 8,350 for volunteers D and E, respectively.

In view of the apparent biphasic nature of the loss in HAV infectivity on fingerpads, infectious virus half-lives and K_i values (expressed as the rate of loss in virus

infectivity in $\log_{10}$ per minute) were determined separately for the 0- to 240-min and the 60- to 240-min periods. The half-lives of the virus for both the periods were the shortest in volunteer E (5.50 and 14.44 h, respectively) and the longest in volunteer D (7.70 and 23.10H, respectively) (Table 1). However, the differences in virus half-lives and K_i values for the five volunteers were not found to be statistically significant either for the 0- to 240-min period or the 60- to 240-min period.

HAV transfer after 20 and 60 min of drying. The mean amounts of virus on the fingerpads and disks at 0 min were 3.1×10^4 and 2.9×10^4 PFU/ml, respectively. There was always virus transfer. A three-way analysis of variance revealed the significant main effects because of the mode of transfer, pressure, and friction ($F = 3.35$, $P < 0.05$; $F = 133.51$, $P < 0.05$; and $F = 81.29$, $P < 0.05$, respectively). A post hoc analysis (Tukey tests) of the main effect of the transfer mode showed a significant difference in virus transfer between the fingerpad-fingerpad (mean, 634.08 PFU) and the disk-fingerpad (mean, 454.72) modes. Similarly, there was a significant difference between the fingerpad-disk (mean, 607.21) and disk-fingerpad modes in the amount of virus transferred. The amount of virus transferred was not significantly different between the fingerpad-fingerpad and the fingerpad-disk modes.

The analysis also revealed significant differences in virus transfer because of pressure and friction. The increase in pressure from 0.2 kg/cm^2 resulted in a significant increase in the amount of virus transferred ($F = 133.51$, $P < 0.05$); the mean transfer rate went from 212.82 PFU at 0.2 kg/cm^2 to 917.85 PFU at 1 kg/cm^2. Similarly, the

incorporation of friction resulted in a significant elevation in the amount of virus transferred ($F = 81.29$, $P < 0.05$). The mean HAV transfer rate with no friction was 290.27 PFU, whereas with friction, the mean value was 840.40 PFU. The combination (interaction) of pressure and friction was also found to significantly affect the amount of HAV transferred ($F = 33.98$, $P < 0.05$). The two-way effect of mixing pressure and friction is illustrated in Fig. 2. The combination of friction and the higher pressure (1 kg/cm^2) resulted in a profound increase in HAV transfer (mean, 1,370.76). No statistically significant interactions were observed between mode of transfer and pressure and friction, mode of transfer and friction, or mode of transfer and pressure.

HAV transfer over a period of 4 h. The ability of HAV to be transferred over a 4-h period was assessed by using two volunteers (volunteers A and E). The three modes of virus transfer were tested by using only the low level of pressure without any friction. The mean amounts of virus at 0 min in the 10-μl inoculum on fingerpads and disks in these experiments were 1.1×10^4 PFU (1×10^4 to 1.2×10^4 PFU) and 1.08×10^4 PFU (1.07×10^4 to 1.09×10^4 PFU), respectively.

(i) Virus transfer from disks to fingerpads. Nearly 33% of the input virus PFU remained detectable on the disks at the end of 4 h under ambient conditions of temperature (22 ± 2°C) and relative humidity (45 ± 5%) (Fig. 3). When a clean fingerpad was pressed against a disk with an inoculum that was dried for 20 min, about 22% of the PFU (2,667 PFU) could be transferred, whereas no detectable virus was transferred to fingerpads when the inoculum was left to dry on the disks for 4 h.

(ii) Virus transfer from fingerpads to disks. Nearly 25% of the input virus PFU remained detectable on the fingerpads even after 4 h (Fig. 4). The amount of virus transferred from contaminated fingerpads to clean disks on contact was nearly 27% of the virus (3,484 PFU) remaining after 20 min of drying; when the inoculum was dried for 4 h, 1.6% of the surviving virus (50 PFU) was transferred.

(iii) Virus transfer between fingerpads. About 10,433 PFU of HAV was deposited on the fingerpad of volunteer E, but only 1,967 PFU (19%) remained at the end of 4 h (Fig. 5). The survival pattern was similar to that observed earlier (Fig. 1). The amount of HAV transferred from the fingerpads of volunteer E to the fingerpads of volunteer A was 2,800 PFU after 20 min of drying (23% of the available PFU) but only 17 PFU (0.9% of the available PFU) at the end of 4 h.

DISCUSSION

Gwaltney et al. (14) demonstrated that as much as 70% of the infectious rhinovirus on hands can be transferred to a recipient finger after a contact of only 10 s. Similarly, Hendley et al. (20) have shown that dried rhinoviruses picked up from environmental surfaces may persist on human hands long enough to permit self-inoculation and that virus transmission may proceed by transfer of the virus from the hands of the infected person to an intermediary surface or directly to the fingers of a susceptible recipient. Respiratory syncytial virus has also been shown to survive in the environment, allowing its transfer to human hands (18), and studies on the transfer of

human rotavirus between hands and inanimate surfaces also implicate hands and inanimate surfaces as potential rotavirus vehicles (2). The results of all of these studies suggest that human hands and environmental surfaces may be potential routes of virus transmission.

The results presented here show that considerable amount of HAV remained infectious on the fingerpads after 4 h, even though 68% of virus infectivity was lost in the first 1 h. Whereas the reasons for the biphasic pattern of virus decay and the relatively rapid drop in virus titer during the first 60 min are not clear, it may be related, at least in part, to the rate of moisture loss from the inoculum. Although HAV inocula appeared dry after about 20 min, the decay curve shown in Fig. 1 leveled off at around 60 min. This suggests that 60 min was the end of the drying period.

Other factors, such as the physiology and chemistry of the skin surface, may also play a role in HAV survival and inactivation on hands. Both immunoglobulins and serum proteins contained in normal human sweat have been shown to inhibit bacterial growth (38). It is not clear that these substances behave in a similar way with viruses. Surface immunoglobulin A has also been shown to contribute to the immunological defense of the skin (23). The presence of two HAV subpopulations in the inocula may also be responsible for the biphasic pattern in virus decay, so that the more susceptible virus population is inactivated faster during the drying period and the more resistant virus population persists over the whole study period (4 h).

Because a fecal suspension of HAV was used to simulate natural conditions in which HAV contaminates both animate and inanimate surfaces, it is expected that the results may be an indication of what may occur in the field. Residual HAV on human hands at the end of 4 h could also be transferred to others and may be enough to initiate infection upon self-inoculation. High levels of infectious HAV were exchanged between the fingerpads and the disks over the 4-h period. These results suggest that human hands and fomites are potential ways in which self-inoculation and communication of the virus can occur over an extended time. The significance of pressure and friction in determining the amount of HAV transferred in the three models examined in this study was shown. We are not aware of any previous quantitative studies in which such an interaction in the transfer of infectious virus has been documented.

Virus transfer between contaminated and clean surfaces is greatest when the inoculum is wet (2, 14, 18, 20) and is observed to decrease as the virus dries. Also, during drying some virus is usually inactivated. In the observed transfer of infectious virus at specific times, the effects of drying on virus inactivation as well as the physical transfer of virus particles and their recovery are confounded. However, in order to assess the virus loss because of drying, we used a reference point, which was the indication that the virus inocula were visibly dry on the contaminated surfaces. An inoculum of 10 μl of HAV in fecal suspension appeared to be dry after 18 to 20 min on the metal disks (33) and 11 to 17 min on fingerpads. For this reason, 20 min was taken as the minimum time that HAV inocula would require to initially become dry. The first

reading was therefore taken at 20 min and indicates virus loss because of drying.

The period of experimentation used in this study is within the limits for human hands to stay contaminated before a hand wash or interaction with other animate and inanimate environments. The metal disks used in this study were selected to represent environmental surfaces. In our previous studies (33), stainless steel disks allowed the virus to persist for several days; glass and plastic surfaces may behave similarly (44).

HAV outbreaks are often associated with hospitals (3, 13, 25, 45). The mode of transmission in those studies was unclear, but the source was usually an asymptomatic child or adult. The observation that the spread of the infection is associated with nurses and other medical personnel suggests that human hands may play a vehicular role in virus transmission.

An assessment of HAV outbreaks in the community shows that diaper-changing personnel in many day-care centers also participate in food-handling activities (9, 16); HAV may be acquired from children who are excreting HAV; the majority of these children are asymptomatic (10). Although no such study has been done for HAV, the day-care center environment has often been found to be contaminated with enteric organisms which have modes of transmission similar to those of HAV (9, 24, 39).

HAV that has contaminated toys and other play items may persist for prolonged periods of time (33); toys are shared items in day-care centers, and children are known to suck them frequently (4, 9). Such behavior tends to encourage virus transmission through the fecal-oral route

among the children. The children also associate closely with the environment around them as well as with adults assigned to them in the day-care center (4). Interactions between such a contaminated environment and the institutionalized population, especially children, may recycle the virus until susceptible hosts, usually adults, are infected. Such a secondary spread of HAV often results in communitywide outbreaks (17).

Restaurant-associated HAV outbreaks are frequently reported (7, 26, 28, 35). In these instances, the index case was frequently a food handler who was subclinically infected with HAV. Secondary spread of the disease is a prominent feature of the outbreaks (7, 26, 28) and may occur over an extended period of time (30), indicating a continuing source of infection. Refrigerated foodstuffs as well as foods stored at room temperature after handling are know to permit the survival of HAV for several days (11) and, together with the index case, furnish a continued source of the virus. It has also been suggested that HAV which persisted on a sandwich board contaminated by hands moistened with oropharyngeal secretions from an anicteric food handler was responsible for contaminating many sandwiches (28).

The potential of human hands to spread viral agents has serious implications in the food industry and eating establishments, and it may be particularly important when foodstuffs do not require cooking after handling. HAV contamination of foodstuffs by an institutional food handler can culminate in communitywide HAV outbreaks (6, 7, 19, 26, 28, 35).

The persistence of HAV on surfaces and the ability of the virus to be interchanged in animate and inanimate environments suggests that human hands and environmental surfaces constitute important epidemiological factors in the spread of HAV. Approximately 50% of HAV cases reported to the Centers for Disease Control every year show no identifiable source of the virus (15). Previously, we have shown that HAV on environmental surfaces can remain infectious for several days (33). Taken together with the results of this study, this suggests that human hands and environmental surfaces may serve as sources of HAV dissemination, especially in institutionalized populations.

Hand washing is probably the single most important measure for controlling and preventing the spread of infections (5, 22, 43). However, compliance with hand washing guidelines among medical personnel is frequently suboptimal (1, 10) and is sometimes ignored altogether (9). Moreover, many commonly available surface disinfectants have been shown to be ineffective against HAV (29), and Mbithi et al. (34) have shown that commonly available antiseptics are inefficient in removing or inactivating HAV. Use of antiseptics has previously failed to contain nosocomial HAV (3, 45), thus permitting secondary spread of the virus in hospital wards.

The findings of this study show that the control and prevention of HAV outbreaks in settings such as day-care centers, hospitals, eating establishments, and other, similar facilities must place a great emphasis on both human hands and environmental surfaces as virus vehicles.

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TRANSMISSION OF EXPERIMENTAL RHINOVIRUS INFECTION BY CONTAMINATED SURFACES

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Gwaltney, J.M., Jr. (Dept. of Internal Medicine, U. of Virginia School of Medicine, Charlottesville, VA 22908) and J. O. Hendley. Transmission of experimental rhinovirus infection by contaminated surfaces. *Am J Epidemiol* 1982;116:828-33.

Transfer of experimental rhinovirus infection by an intermediary environmental surface was examined in healthy young adults, in four studies done in 1980-1981, by having recipients handle surfaces previously contaminated by infected donors. Recipients touched their nasal and conjunctival mucosa after touching the surfaces. Five (50%) of 10 recipients developed infection after exposure to virus-contaminated coffee cup handles and nine (56%) of 16 became infected after exposure to contaminated plastic tiles. Spraying of contaminated tiles with a commercially available phenol/alcohol disinfectant reduced (p=0.003) the rate of recovery of virus from the tiles from 42% (20/47) to 8% (2/26). Similarly, the rate of detection of virus on fingers touching the tiles was reduced (p=0.001) from 61% (28/46) with unsprayed tiles to 21% (11/53) with sprayed tiles. Fifty-six percent (9/16) of the recipients exposed on three consecutive days to untreated tiles became infected while 35% (7/20) touching only sprayed tiles became infected with rhinovirus (p=0.3). These studies indicate

that experimental rhinovirus colds can be spread by way of contaminated environmental surfaces and suggest that disinfectant treatment of such surfaces may reduce risk of viral transmission by this route.

acute respiratory disease viruses; disinfectants; rhinoviruses

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Rhinovirus may infect susceptible persons by means of self-inoculation of virus which accidentally contaminates the fingertips (1-3). It is clear that virus on the fingers of volunteers is efficiently transferred by contact to the nasal and conjunctival mucosa where it routinely produces infection (2). An unsettled question is how virus might get onto the fingertips of well persons. The two most likely sources are skin of persons with colds and objects in the environment which have been contaminated with virus by ill people.

Rhinovirus has been routinely recovered from the hands of persons with both natural (1) and experimental (2, 4, 5) infections. Also, rhinovirus was detected on 6 percent of selected objects in the homes of persons with rhinovirus colds (2). Thus, measures which would eliminate or reduce viral contamination of skin and environmental surfaces may be useful in interrupting viral spread and controlling rhinovirus colds.

Hand treatment with a virucidal preparation was successful in interrupting rhinovirus transmission between volunteers (6), and its effectivenesses is now being studied under natural conditions. The current study was done to determine if rhinovirus colds could be efficiently transmitted in volunteers by way of contaminated surfaces. In

addition, the effectiveness of a commercial disinfectant in killing virus on surfaces and interrupting transmission at that point was assessed. Surfaces contaminated with virus by volunteers with experimental colds were sprayed with phenol/alcohol disinfectant or left unsprayed to determine the efficacy of this form of disinfection in interrupting spread of infection to volunteers touching the surfaces.

MATERIALS AND METHODS

Volunteers

Healthy, young adults (≥18 years) were recruited from the University of Virginia community, Charlottesville, VA, in four studies done in 1980-1981. Volunteers were selected if serum-neutralizing antibody titers to the challenge virus were low or absent (≤2). Volunteers who had symptoms of a respiratory illness in the week prior to the study were excluded. Women who were pregnant or not using accepted contraceptive methods were excluded. The experimental protocol was approved by the University of Virginia Human Investigation Committee and volunteers gave written consent to participate in the study.

Virus challenge pool

A pool of a presently unnumbered rhinovirus strain (HH) in nasal secretions from a young adult with a rhinovirus cold was used for the challenge inoculum. Nasal secretions were diluted 1:5 in Hanks' balanced salt solution before storage in multiple aliquots at -70 C. The titer of the pool was 10^3 tissue culture infective $dose_{50}$ ($TCID_{50}$)/

ml. The pool had been tested for and found free of other viruses, pathogenic bacteria, nycoplasmas, fungi, and *Mycobacterium tuberculosis*. The individual from whom the pool was obtained was otherwise in good health and had a negative chest x-ray, tuberculin skin test, and serum test for hepatitis B virus.

Specimen collection and testing

Samples of nasal and pharyngeal secretions were obtained by cotton swabs which were placed in 5 ml of beef heart infusion broth with 1 per cent bovine serum albumin and antibiotics. Fingers were rinsed with 1.5 ml of the same broth without antibiotics, and the rinse was combined with 1 ml of broth with antibiotics before testing. The plastic and ceramic surfaces were cultured by rinsing with 2.5 ml of the collecting broth with antibiotics.

Viral culture was performed by standard methods (7) in human embryonic lung cells (WI-38) and/or fetal tonsil cells in screw-capped tubes. Serum specimens were obtained before and three weeks after challenge. Serum-neutralizing antibody titers were determined against the challenge virus by a standard method (7).

Infectivity and illness criteria

Infection was judged to have occurred if virus was recovered from a nose/throat culture an/or if there was a fourfold or greater serum neutralizing antibody rise to that challenge virus. Determination of the occurrence of a symptomatic illness was based on a modification of the

method of Jackson et al. (8) Illness was considered present if a volunteer had a minimum symptom score of six and had the subjective impression that he had a cold, or if rhinorrhea was present on three or more days of observation.

Disinfectant

A commercial spray disinfectant (Lysol spray disinfectant, Lehn and Fink Products Group, Sterling Drug Inc., Montvale, NJ) containing the active ingredients O-phenylphenol (0.1 percent), N-alkyl (C_{18}, 92 percent; C_{16}, 8 percent), N-ethyl morpholinium ethylsulfates (0.035 percent), and alcohol (67.748 percent) was dispensed from an aerosol can as spray.

Experimental plan

Infection of donors. Immediately following a nasal wash with normal saline (5 ml/nostril), donors were inoculated intranasally with 0.25 ml per nostril of a $10^{0.5}$ dilution of the virus pool. Challenge was done on two successive days. Donors were placed in isolation 12 hours after the second inoculation (the earliest that infected donors would be expected to shed virus). They were monitored on three exposure days (days 3, 4, and 5 after the first challenge) for the amount of virus in their nasal secretions and on their hands (after they had contact with the contaminated surface). Each donor was lodged in an individual room and separated from the recipients at all times.

Donor handling of surfaces. Symptomatic donors deliberately contaminated their hands as in nose blowing and nose wiping immediately after contact with a ceramic coffee cup or a 12.5 x 12.5 cm square plastic tile of the type used for counter tops. The donors then held the handle of the coffee cup or rubbed the tile with the fingertips of each hand for approximately five seconds. This was repeated on three consecutive exposure days, beginning on the third day after the donor's first inoculation. The same donor was not necessarily paired with a particular recipient on each of the three exposures days.

Treatment of tiles. Ten minutes after the donors handled the surfaces of the tiles, the tiles were sprayed with disinfectant or not. The dispenser was held 15-20 cm from the surface which was sprayed for two to three seconds until covered with mist.

Recipient handling of contaminated objects. For exposure to the coffee cups, the recipients held and rubbed the handle of the cups as if drinking. For exposure to the tiles, the recipients touched and rubbed the surfaces with the fingertips. This was done 10 minutes after the spray treatment to allow time for treated tiles to dry. Fingertips of each hand touched a different tile (of the two contaminated by the donor). After contact with coffee cup or tiles, the recipient placed the fingers in contact with the conjunctival and nasal mucosa as in rubbing the eyes and probing the nose. The recipient's fingers and coffee cup handles or tiles were then sampled for virus. Recipients were housed in isolation in individual rooms beginning 12 hours after the second contact.

RESULTS

Exposure by coffee cup handles. Three donors were used to contaminated coffee cup handles. Five to the 10 recipients exposed to the coffee cup handles became infected. Virus was recovered in nose/throat specimens from all five infected recipients on two or more days, and all had fourfold or greater serum-neutralizing antibody rises to the challenge virus. Virus was recovered from one coffee cup handle specimen and from hand rinse specimens of two of the infected recipients.

Exposure to plastic tiles. In four experiments, recipients contacted tiles which had been contaminated by donors and either sprayed with disinfectant or left unsprayed (table 1). Virus was detected on 20 (42.5 percent) of 47 tiles that received no treatment compared to 2 (8 percent) of 26 which had been sprayed with disinfectant ($p= 0.003$). Virus was detected in 28 (61 percent) of 46 hand rinses of recipients in the no treatment group and 11 (21 percent) of 53 hand rinses of recipients in the disinfectant spray group ($p=0.001$). Nine (56 percent) of 16 recipients who contacted tiles which had not been sprayed became infected. Seven (35 percent) of 20 recipients became infected after touching tiles which had been sprayed with disinfectant ($p=0.3$). All infected recipients had virus in their nose/throat cultures on one or more days, and 13 of 16 had fourfold or greater rises of serum-neutralizing antibody titer to the challenge virus.

Results in both the treatment and no treatment groups were combined and analyzed to determine whether the presence of detectable virus on the tiles correlated with viral presence on the recipients' fingers and whether the

virus on the recipients' fingers correlated with the development of infection. In 15 (75 percent) of the 20 instances in which tile cultures were virus-positive, fingers of recipients contacting the tiles were positive for virus. In contrast, only 18 (36 percent) of the 50 instances in which tile cultures were negative were the matching hand cultures from recipients positive ($p=0.007$, Fisher's exact test). Virus was detected on the hands of 23 recipients at least once during the three exposure days. Fourteen (61 percent) of these 23 individuals were infected, whereas only 2 (15 percent) of 13 recipients whose hand cultures were negative became infected ($p=0.02$, Fisher's exact test). Thus, it appeared that the presence of detectable virus was a controlling variable both in viral transfer from tile to hands and from hands to susceptible mucosa.

Clinical illness in recipients

Of the 16 recipients who became infected, 10 (62.5 percent) met the criteria for illness. Symptom scores of these individuals ranged from 8 to 42 with a median score of 23. The illness had the characteristics of a typical rhinovirus cold.

DISCUSSION

In this study, experimental rhinovirus infection was transmitted by two types of contaminated surfaces, ceramic coffee cup handles and plastic tiles. The efficiency of transmission by the two surfaces was similar, 50 percent for the coffee cups and 56 percent for the tiles. Treatment of the tiles with a phenol/alcohol spray disinfectant was effective in reducing viral contamination of

the tiles and viral soiling of fingers having contact with the tiles. Further, the infection rate of susceptible recipients contacting the tiles may have been reduced by the disinfectant treatment, although the difference was not statistically significant. Since we found that the risk of acquiring infection was directly related to the presence of viral contamination of the fingers and this, in turn, was related to the presence of virus on the tiles, it could reasonably be expected that the use of a disinfectant which would eradicate virus on surfaces would reduce the infection rate by this route. In view of this, it was felt that further use of volunteers to investigate this question was not indicated.

The question of whether disinfection of environmental surfaces would result in reduced transmission of rhinovirus colds under natural conditions cannot be answered until the importance of the hand contamination/self-inoculation route of spread is known. At present, the evidence with experimental colds suggest that the hand/self-inoculation route is likely to be important under natural conditions (2,3), but direct evidence bearing on this question has not been obtained. The disinfectant tested in these experiments was moderately effective in eliminating rhinovirus from a surface and has proven to be safe through long use. However, it wasn't ideal in that it did not completely eliminate viral contamination of the tiles and one-third of the recipients in the tile-treated group became infected. If the hand/self-inoculation route does prove to be important, environmental disinfection may have a useful role in controlling colds.

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Letter to the Editor

Inactivation of Human Immunodeficiency Virus-1 at Short Time Intervals Using Undiluted Bleach

To the Editor: Several modes of transmission are implicated in the spread of human immunodeficiency virus-1 (HIV-1) and acquired immunodeficiency syndrome (AIDS) including injection practices, blood transfusion, sexual activity, and perinatal transmission. Injection drug users are at risk for HIV-1 transmission due to sharing needles (1-3). Health care workers and laboratory personnel are at risk for HIV-1 infection through accidental needle stick injuries and exposure of their mucous membranes to HIV-1 contaminated fluids while providing treatment to infected patients or processing fluids and tissues from these individuals (4).

The use of hypochlorite-based household bleach has been widely recommended to health care workers and laboratory personnel as a means to inactivate HIV-1 (5,6) and injection drug users have been advised to clean their injection equipment with household bleach (7-9).

Prior studies have characterized the use of hypochlorite and other reagents to inactivate HIV-1 (10,11). Our investigation provides additional detailed information regarding the shortest exposure time required to inactivate HIV-1 with undiluted household bleach.

METHODS

Normal peripheral blood mononuclear cells (PBMNCs), cells infected with HIV-1, and supernatant stocks of HIV-1 were prepared according to standard methods (12,14). HIV p24 antigen was detected utilizing kits from Abbott Laboratories (Chicago, IL. U.S.A), according to manufacturer instructions. The p24 assay is generally acknowledged to be more sensitive than reverse transcriptase assays (15). Household bleach was purchased from a local supermarket and contained 5.25% sodium hypochlorite.

HIV-1 was pelleted from infected cell culture supernatants at 13,000 relative centrifugal force (rcf) (12) in microcentrifuge tubes. The pellets were then exposed to bleach for various periods of time, ranging from 15 s to 5 min. After gently removing the bleach from the tubes, the pellets were suspended immediately in cell culture medium and used to infect normal donor PBMNCs using standard techniques (13,14). The carryover of bleach into the culture medium from pelleted virus was approximately 0.2% (v/v), which we determined to be completely nontoxic for PBMNC cultures. Toxicity only occurs when the bleach concentration is above 1% (v/v) (data not shown). The PBMNC cultures were maintained for up to 28 days and were assayed for virus production at weekly intervals by determination of HIV-1 p24 is the culture medium. Cultures were classified as negative if all determinations of p24 production were below 5 pg/ml for that culture. A culture was determined to be positive when p24 production was three times above the maximum background value of 5pg/ml. The peak mean value was reported for these cultures and generally occurred

after 14 or 21 days of cell culture. All experiments were done in triplicate, and exposures of virus to bleach for less than 1 min were repeated in triplicate two times.

The viability of normal PBMNCs was determined after treatment with bleach by typan blue dye exclusion and taking counts using hemacytometer. Aliquots of 10 million cells were pelleted by centrifugation at 900 rcf and suspended in 0.5 ml of undiluted bleach. After exposure to bleach ro 15 to 60 s the suspensions of cells were immediately diluted to 50 ml (100-fold) using RPMI medium containing 500 units/ml each of penicillin and streptomycin, and 1% of fetal calf serum. A 1% dilution of bleach and lower concentrations was completely harmless for cell viability as stated above. The cells were then pelleted as above and suspended in 0.5 ml of RPMI medium supplemented with antibiotics and 20% fetal calf serum. Cell viability was determined in duplicate for each time point up to 60 s and after maintenance in cell culture medium for 18 h.

RESULTS

HIV-1 was completely and consistently inactivated by undiluted household bleach at all tested exposures of 30 s or longer. As summarized in Table 1, HIV-1 was not completely inactivated at shorter intervals or exposure to bleach. The p24 antigen concentrations reported in Table 1 are averages for triplicate experiments at the indicated bleach exposure time periods. HIV-1 was not consistently inactivated at exposures to undiluted bleach of 15 and 25 s. Exposure of HIV-1 to a 10% dilution of bleach, using

the same methodology, did not inactivate the virus even at 5 min of exposure.

PBMNCs were also shown to be rapidly fragmented and killed in the presence of undiluted bleach. There was complete loss of viability at 25 but not at 20 s of exposure to undiluted bleach (Table 2). With 10% diluted bleach, 4% of the PBMNCs remained viable after 60 s. After 18 h in cell culture medium, 3% of the cells exposed to 10% diluted bleach remained viable.

DISCUSSION

Our present investigation gives a precise measurement of bleach exposure time for the inactivation of HIV-1. The methods used demonstrate that exposure of pelleted virus to undiluted household bleach inactivates HIV-1 and kills PBMNCs within a short exposure period, but a 10% dilution of bleach is ineffective at considerably longer exposure periods. We found that the 10% dilution had viable virus at 5 min of exposure. Based on these results, 10% diluted bleach does not appear to have any practical, effective value in the inactivation of HIV-1.

There are clear implications from these findings: To inactivate HIV-1, use undiluted bleach for a minimum exposure of 30s. It is also important to note that PBMNCs were killed after 25 s of exposure to bleach.

It is extremely important to make this information available to all persons at potential risk of exposure to HIV-1, including health care and research workers as well as injecting drug users. This is crucial as part of national and international efforts to halt the spread of AIDS.

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Thirty-nine products representing six categories of disinfectants (alcohols, chlorines, dilute glutaraldehydes, iodophors, phenolics, and quaternary ammonium compounds) were first tested in the absence of bioburden, using four test methods with five test organisms. Products that performed best were retested with the same methods and organisms in the presence of both serum and whole blood, using 3- and 10-minute contact times. Only products containing high ethyl alcohol had consistently high antimicrobial activity regardless of the test method, test organism, or contact time used both in the absence and presence of bioburden. Although these specific formulations demonstrated ability to penetrate and inactivate high concentrations of microorganisms within heavy bioburden, optimum disinfection of environmental surfaces is highly formulation dependent. Other products tested showed deficiencies that contraindicate their use as environmental surface disinfectants in clinical dental settings.

Antimicrobial activity of environmental surface disinfectants in the absence and presence of bioburden

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Current methods of dental treatment cause widespread contamination of the operating area.[1-8] Droplet

splatter, flying debris, contaminated hands and instruments, and operation of automated instruments such as handpieces, ultrasonic scalers, and air-slurry polishers contribute to the problem.[1-3,9-20]

The potential of contaminated environmental surfaces to transmit infection continues to be questioned by some investigators. However, confirmation of long-term survival of large numbers of pathogenic organisms on a variety of surfaces[21-35] has made infection via contaminated surfaces credible, and experimental data corroborate the possibility. For example, both rotavirus and rhinovirus have been transferred from contaminated hands, to objects, and to clean hands in sufficient numbers to elicit infection in susceptible humans.[36-37]

Furthermore, colds caused by rhinovirus have been transmitted to human volunteers experimentally via contaminated ceramic cup handles and plastic tiles.[38] Comparable correlations at the clinical level have been difficult because so much time elapses between infection and overt symptoms that subjects often cannot recall objects contacted. Even when recall is possible, the incubation time lapse can exceed organism survival time on inanimate surfaces, and culturing becomes impossible. Therefore, clinical reports linking infection with objects have been based on circumstantial evidence. Examples include epidemiologically linked diseases,[21,39-44] spread of infection in facilities in which direct contact of infected subjects was impossible,[45-46] clinical experiments,[47] and documentaries.[48] Reports such as these make it impossible to rule out environmental surfaces as fomites.

Until data become available to demonstrate conclusively that contaminated environmental surfaces cannot transmit infections, clinicians cannot ignore or treat them lightly. As dental operatories have innumerable environmental surfaces that are contaminated during routine patient treatment, the effectiveness of products used to disinfect these surfaces must be examined. This investigation was conducted to test a number of commercial products to identify those with broad-spectrum, rapid antimicrobial activity both in the absence and presence of bioburden.

Methods and materials

The general protocol specified use of four test methods and five test organisms with 39 disinfectants in the absence of bioburden. Disinfectants demonstrating best antimicrobial activity under these conditions were then tested with the same methods and organisms in the presence of bioburden.

Disinfectant selection and preparation

Table 1 lists the disinfectants selected for this study based on a product-use survey,[49] manufacturers' communications, and literature review. All products were prepared according to manufacturers' directions immediately before testing. When dilution was specified, sterile deionized water was used.

Test organism selection and preparation

Test organisms were: *Pseudomonas aeruginosa* ATCC 15442, *Salmonella choleraesuis* ATCC 10708, *Staphylococcus aureus* ATCC 6538, *Mycobacterium bovis* (BCG) ATCC 35743, and poliovirus type I (Mahoney strain). The four bacteria were selected because they are specified as test organisms by the Environmental Protection Agency (EPA) to register hospital disinfectants[50] and to establish tuberculocidal claims.[51] Poliovirus I was selected because it is resistant to inactivation by many disinfectants.[52-54]

Bacteria were prepared according to EPA specifications, except stock cultures were stored in liquid nitrogen. Poliovirus was grown in HeLa cells, harvested by multiple freeze/thaw cycles and cesium chloride (CsCl) banding, and stored at 4 C in CsCl.

Test methods

Association of Official Analytical Chemists Use Dilution Method (AOAC UDM). The standard AOAC UDM, using 60 stainless steel penicylinders per replicate, was performed precisely as described in AOAC literature.[50] Secondary subculture was performed on each carrier and both subcultures were incubated at 37 C for 48 hours. For bioburden testing, the same method was used except penicylinders were coated with 50:50 vol/vol mixture of human whole blood and bacterial culture.

Environmental Protection Agency Tuberculocidal Activity Test Method (EPA TB ATM). The standard quantitative EPA TB ATM was performed precisely as specified in EPA literature.[51] For bioburden testing, the same method was

used except test suspensions contained 50% horse serum which delivered a 5% concentration of horse serum to the disinfectant.

Virucidal Suspension Test. A suspension test, rather than a carrier method, was used for all virucidal testing because all attempted procedures for drying virus onto carrier surfaces, and subsequent virus recovery, caused loss of viral titer that was unpredictable and unacceptably high (>2 $\log_{10}$ units). Tests performed without blood used 100 µL of virus, $\geq 10^6$ plaque-forming units (PFU), added to 900 µL of disinfectant. After prescribed contact times, phosphate-buffered saline solution was used for serial 1:10 dilutions, and four consecutive dilutions of virus were assayed in duplicate on monolayers of HeLa cells for infectious poliovirus. Twelve-well plates (12-Well Tissue Culture Cluster 3512, Costar) were incubated at 37 C for 48 hours and stained. Log_{10} reductions were calculated from plaque counts. Disinfectants yielding three $\log_{10}$ reductions in virus titer were evaluated for interference with virus attachment to HeLa cells. Each 12-well plate included controls to assay for titer of viral challenge and test for system contaminants. Also, disinfectant cytotoxicity to HeLa cells was determined. In cases in which cytotoxicity was detected, dilution was used to eliminate this effect. Dilution was also used to eliminate the effect of residual disinfectant on the virus in the assay system.

Testing with human whole blood used the same procedures described except 10µL of poliovirus ($\geq 10^6$ PFU) was added to 100 µL of blood and allowed to stand for 1 minute before 900 µL of disinfectant was added. Controls were included to determine amount of virus inactivated by blood. For tests in which urea was included to disrupt

ethyl alcohol-induced blood aggregates, the same procedures were employed except that siliconized tubes (Sigmacote, Sigma Chemical) were used and 9.0 mL of 7.0 mol/L urea was added to the blood-virus-disinfectant mixture after prescribed contact times. Log_{10} reductions were calculated for all work involving poliovirus (both with and without bioburden) using the formula: log_{10} reduction = log_{10} (titer of viral challenge per mL) – log_{10} (titer infectious virus per mL after exposure to disinfectant).

Clinical Research Associates Environmental Wipe Method (CRA EWM). A test was devised to mimic dental clinical procedures used for disinfection of environmental surfaces to determine if wiping with disinfectant-soaked gauze sponges inactivated organisms dried onto surfaces, both in the absence and presence of bioburden. Silicone adhesive (Mirror 3 Tray Adhesive, Kerr/Sybron) and caulking (Silicone II GE5070, General Electric Co) were used to attach an $8^{1/16-}$ x $11^{7/8-}$ in piece of laminated plastic counter covering (1595-6 Black Wilsonart, Ralph Wilson Plastic Co) to $8^{1/4-}$ x 12-inch polypropylene trays (Size B Trays 20Z401, Zirc Dental), which were trimmed to fit snugly under their polyethylene lids (Tray Cover 20Z441, Zirc Dental). Figure 1 shows the test tray after construction and before inoculation with test organisms. Lids and trays were sterilized with ethylene oxide (ETO) for 2 hours at 135 F and aerated for 8 hours before 2 mL of bacterial suspension was applied with a sterile 2- x 2-in cotton filled gauze sponge (Cotton Filled Sponges 6000207, Healthco). After the organisms applied to the trays were completely dried (20-30 minutes) in a laminar

flow hood (NU-408FM-600, Nuaire), 3.5 mL of disinfectant was pipetted onto a sterile gauze sponge which was then used to wipe the test surface for 10 seconds using about 150-g pressure with overlapping strokes (20 left to right, followed by 20 top to bottom). Disinfectants sold in aerosol spray cans were treated in the same manner to standardize the amount of disinfectant delivered to the contaminated surface by spraying the disinfectant into a sterile test tube before pipetting. After wiping, the disinfectants were left on the trays for 3 minutes before one of two methods was used to determine the number of viable organisms remaining on the tray. The method depended on the test organism.

M bovis coated trays were flooded with 50 mL of tryptic soy broth (TSB) with neutralizers (ingredients are enumerated in section on media and neutralizers), and scrubbed for 1 minute with a sterile polypropylene brush (Nail Brush 501, Kellogg Brush Co) to remove and suspend viable organisms. The fluid was collected and diluted, and duplicate 1-mL samples of each dilution, plus the undiluted fluid (approximately 40 mL), were passed through a 0.45-µm filters. Each filter was washed twice with 100 mL of sterile 0.1% peptone water, placed on Mycobacterium 7Hll agar, and incubated at 37 C for 21 days. To allow calculation of $\log_{10}$ reductions, trays wiped with water were included in all tests. Log_{10} reductions were calculated using the formula: $\log_{10}$ reduction = $\log_{10}$ (number of viable organisms from water control tray) – $\log_{10}$ (number of viable organisms from test tray).

P aeruginosa, S choleraesuis, and *S aureus* remaining viable organisms were assayed directly on the trays by adding 300 mL of tryptic soy agar (TSA) with neutralizers

at 45 C. Trays were covered with their lids and incubated at 37 C for 48 hours. Colonies were counted, with > 500 designated as "too numerous to count" (TNC). Trays wiped with water were included as organism viability controls. Results of this testing were reported two ways: by the number of colony forming units (CFU) for each of the three test bacteria that survived disinfectant treatment, and as percent of tests less than TNC. To determine the percent of tests less than TNC for a particular disinfectant, the following formula was used: percent of tests less than TNC = number of tests less than TNC for the three organisms ÷ total number of tests x 100. To determine the mean percent of tests less than TNC for a category of disinfectants with the same active ingredient, the mean of all disinfectants within the category was calculated.

The CRA EWM bioburden testing with all four bacteria used the same methods, except cultures were mixed 50:50 vol/vol with human whole blood; 1 mL of this mixture was spread on trays with a sterile glass rod. For tests with *S aureus* and *M bovis* in which urea was included to disrupt blood aggregates, the procedures described previously for *M bovis* were used except that 50 mL of 7.0 mol/L urea was substituted for TSB used to suspend organisms from the surface.

Media and neutralizers

For *M bovis*, TSB (Difco) containing 1% Tween 80 (Fisher Scientific), 1% lecithin (Sigma), and 0.4% sodium thiosulfate (Sigma) were used in CRA EWM tests, and Mycobacterium 7H11 agar (Difco) was used for all subcultures.

For *P aeruginosa, S choleraesuis,* and *S aureus,* TSA (TSB + 1.5% Bacto-agar, Difco) containing 0.5% Tween 80, 0.1% lecithin, and 0.1% sodium thiosulfate was used as the subculture medium. Minimum Essential Medium (Irvine Scientific) supplemented with 10% calf bovine serum enriched with iron (HyClone) was used for HeLa cell cultures.

Selection of bioburden and procedures used for testing with bioburden

Because of its clinical relevance, human whole blood was used as the bioburden challenge whenever possible. A concentration of 50% human whole blood in the blood-bacterial suspension mixture was used with the AOAC UDM and CRA EWM tests performed with *P aeruginosa, S choleraesuis,* and *S aureus.* A 10% concentration of human whole blood was used with virus suspension tests because higher concentrations precluded proper mixing and pipetting with some disinfectants. Two different types of bioburden were used with *M bovis* to accommodate test method differences. A 50% concentration of human whole blood in the bacterial suspension was used in the CRA EWM tests with this organism, and 5% horse serum in the bacterial suspension-disinfectant mixture was used in the EPA TB ATM tests. Five percent horse serum was used because it has been specified as the bioburden for other standard EPA tests.[55]

Use of human whole blood as the bioburden required special procedures when ethyl alcohol-containing disinfectants were used because they caused the organism-media-whole blood mixture to form aggregates. Different

chemicals were sought to disperse the aggregates without affecting viable organisms, and 7.0 mol/L urea met the criteria best.[56] Although 7.0mol/L urea destroyed gram-negative bacteria, it allowed almost complete recovery of *M bovis, S aureus,* and poliovirus. Therefore, when ethyl alcohol disinfectants were tested with whole blood bioburden, the CRA EWM tests using *S aureus* and *M bovis* and suspension tests using poliovirus included the addition of 50mL and 9mL of 7.0 mol/L urea, respectively, after disinfectant treatment. The same tests were performed without urea so results could be compared.

Selection of wipe material

As it has been reported that cotton may interfere with the antimicrobial activity of iodophors,[8] this was also evaluated. For this test, sterile GSA centrifuge bottles containing 64 mL each of Biocide and Wescodyne iodophors diluted 1:213 received 8 g of three different wipe materials (Cotton-Filled Gauze Sponges by Healthco, Nu-Gauze rayon/polyester sponges by Johnson and Johnson, and Viva Paper Towels by Scott Paper Co.) After 10 minutes, samples were centrifuged for 20 minutes at 6,000 x g. An aliquot of 9.9 mL was removed from each GSA bottle and 0.1 mL of *S aureus* culture was added. Assays for viable organisms were performed at 3 minutes.

Tests for chemical interference and neutralizer efficacy

To assay for possible toxic effects of ETO residuals or materials, or both, used in the CRA EWM, 300 mL of TSA with neutralizers containing about 100 CFU was poured

into an ETO sterilized CRA EWM tray, a CRA EWM tray disinfected with 70% vol/vol denatured ethyl alcohol and not ETO sterilized previously, and several large sterile petri plates (150 x 15 mm). Tests used three replications each of *P aeruginosa, S choleraesuis,* and *S aureus.*

To test efficacy of the neutralizers in TSB, 1 mL of each disinfectant was added to 9 mL of TSB with neutralizers. After 1 minute, about 100 CFU of *M bovis* was added. Thirty minutes later, the suspension was filtered and subcultured on Mycobacterium 7H11 agar. Efficacy of the neutralizers used in TSA was evaluated by wiping CRA EWM trays with disinfectant, waiting 3 minutes, then filling the trays with TSA plus neutralizers containing about 100 CFU.

Reproducibility of the wipe test

To determine reproducibility of the mean organism challenges applied to CRA EWM test trays both without and with blood, results from water control trays were examined. Reproducibility of CRA EWM test procedures was examined when two different technicians performed the test with *M bovis* mixed 50:50 vol/vol with human whole blood; diluted Clorox (1:5) was the disinfectant. Tests were performed in parallel, and the technicians alternated tray treatment to correct as much as possible for time.

Results

Comparison of data from four different test methods

Figure 2 includes the data from all tests performed in the absence of bioburden. Disinfectants are grouped by main active ingredient to condense the data to facilitate comparison of results obtained with the four different test methods (AOAC UDM, EPA TB ATM, CRA EWM, and suspension tests). The data show a close correlation of results obtained with the EPA-specified test methods (EPA TB ATM and AOAC UDM) compared with the wipe test method (CRA EWM). Significant differences were evident only with iodophors tested against *M bovis*. With this particular disinfectant-test organism combination, the suspension test method (EPA TB ATM) was significantly more permissive than the surface wipe test method (CRA EWM).

Antimicrobial activity in the absence of bioburden

Table 2 lists the detailed data by CFU and PFU under each of the five test organisms for all 39 disinfectants tested in the absence of bioburden. When these data were combined under the primary active ingredient of each disinfectant (Fig 2), it became apparent that overall, in the absence of bioburden, ethyl alcohols and chlorines provided best inactivation of all five test organisms, regardless of the test method or contact time used. Iodophors had intermediate activity. Although they performed well against the virus, they failed to kill *M bovis* dried on plastic laminate surfaces, and they had low activity against the three EPA-specified bacteria. Isopropyl alcohol, alcohol mixtures, dilute glutaraldehyde, phenolics,

and quaternary ammonium compounds all failed to inactivate poliovirus, regardless of the contact time used. The glutaraldehyde and quaternary ammonium compounds also failed to kill the TB organism.

These data also showed: (1) disinfectants could kill the three EPA-specified organisms using the 10-minute EPA specified test (AOAC UDM) and not inactivate resistant organisms with clinical significance such as TB (dilute glutaraldehyde and quaternary ammonium compounds) and poliovirus (isopropyl alcohol, isopropylethyl alcohol mixtures, dilute glutaraldehydes, phenolics, and quaternary ammonium compounds); (2) disinfectants that killed the TB organism did not always inactivate poliovirus (isopropyl alcohols, isopropylethyl alcohol mixtures, and phenolics); (3) poliovirus was resistant to inactivation by several types of disinfectants regardless of contact time (isopropyl alcohol, dilute glutaraldehyde, phenolics, and quaternary ammonium compounds).

Figure 3 shows the performance of each of the 39 disinfectants that were included in the means reported in Figure 2. The data range within each of the eight disinfectant categories is also apparent.

Antimicrobial activity in the presence of bioburden

Table 3 shows results of CRA EWM and suspension tests performed on the 11 disinfectants selected for testing in the presence of bioburden. These data illustrate the adverse effect of whole blood on disinfectant antimicrobial activity. Tests with eight of the 11 disinfectants produced TNC counts when blood was added to the cultures. Only Citrace, Lysol sprays, and 70% vol/vol

denatured ethyl alcohol had consistently high antimicrobial activity across all five test organisms – both in the absence and presence of bioburden. Figure 4 gives a graphic representation of the data in Table 3.

In Figure 5, the 11 disinfectants listed in Table 3 have been grouped by active ingredient to display effects of the different types and concentrations of bioburden (10% and 50% human whole blood and 5% horse serum) used with four different test methods. Overall, the ethyl alcohol category performed best regardless of test method, type or concentration of bioburden, or test organism used. The chlorine category showed high activity against poliovirus, but activity against *M bovis* and *S aureus* was dependent on type and concentration of bioburden used. Whole blood (50%) caused a significant decrease in antimicrobial activity of chlorines, whereas horse serum (5%) did not interfere with antimicrobial activity. Both brands of iodophor had very low activity across all five test organisms in the presence of all types of bioburden. Figure 6 shows the appearance of CRA EWM trays and the EPA TB ATM filters after treatment with Lysol sprays, and Biocide and Wescodyne iodophors.

Figure 7 shows results of tests performed with and without 7.0 mol/L urea, which was used to dissociate aggregates formed when ethyl alcohol products interacted with whole blood bioburden. Separate assays were performed both with the aggregates intact and after dissociation by urea. The goal was to determine if $\log_{10}$ reductions were caused by disinfectant kill or entrapment of viable organisms within the aggregates. The data in Figure 7 indicate entrapment of viable organisms was not generally a problem. Overall, $\log_{10}$ reductions after urea

treatment were equal to or higher than tests not including urea, indicating that these ethyl alcohol disinfectants penetrated the whole blood and inactivated the organisms within.

Antimicrobial activity related to contact time

Figure 8 shows effects of 3-minute versus 10-minute disinfectant contact times in the absence and presence of human whole blood. Generally, increasing contact time made little or no difference in the antimicrobial activity of the four disinfectants tested. Both Biocide and Wescodyne iodophors had almost no disinfectant activity in the presence of blood, even when 10-minute contact times were used. On the other hand, Citrace and Lysol sprays produced greater than 3 $\log_{10}$ reduction for all five test organisms both at 3- and 10-minute contact times and in the absence and presence of whole blood. Therefore, Citrace and Lysol spray were selected for testing at shorter contact times of 2 and 1 minutes.

Figure 9 shows the rapid antimicrobial activity of Citrace and Lysol sprays. In the absence of whole blood, they produced $\geq \log_{10}$ reduction of all three test organisms in 1 minute. With whole blood present, both disinfectants produced $\geq 2.8 \log_{10}$ reduction in 1 minute. With increased contact time, a general increase in kill was achieved.

Antimicrobial activity related to wipe material

Figure 10 shows results from tests of the hypothesis that cotton in gauze sponges used to wipe surfaces interferes

with the antimicrobial activity of iodophors.[8] These data show that iodophor antimicrobial activity was inhibited by paper towels, but cotton and rayon/polyester had no adverse effect at the 3- minute contact time.

Tests for chemical interference and neutralizer efficacy

Table 4 shows results of work performed to test for chemical interference of residuals from ETO sterilization and/or materials used to construct trays used in the CRA EWM. Colony counts of the three test organisms showed inhibition of less than 6 CFU compared with control counts, which indicates that no toxic effects resulted from either variable.

Table 5 reports on efficacy of the neutralizers used in the CRA EWM procedure. The data show adequate neutralization of all products except quaternary ammonium compounds. Although residual activity was present to a small degree with quaternary ammonium compounds, it was not considered a problem because even with this advantage, these products failed to inactivate *M bovis* in the absence of bioburden.

Reproducibility of the wipe test

Reproducibility of the viable organism challenge on CRA EWM rays was demonstrated by the similarity of numbers of organisms computed from water control trays. The mean $\log_{10}$ challenges of 52 (without blood) and 34 (with blood) TB water control trays were 6.01 ± 0.22 and 6.57 ± 0.15, respectively.

Table 6 shows results of reproducibility tests performed on the CRA EWM. Average $\log_{10}$ reductions for *M bovis* achieved by two technicians using Clorox 1:5 in three test replications are shown. These data were consistent both within and between technicians.

Discussion

The goal of this investigation was to identify environmental surface disinfectants that had broad-spectrum antimicrobial activity, rapid action, and effectiveness both in the absence and presence of bioburden. Only Citrace, Lysol sprays, and 70% vol/vol denatured ethyl alcohol met the criteria, regardless of the test method or contact time used. Chemically, the two commercial products are similar. Citrace contains 66.6% wt/wt denatured ethyl alcohol (SDA-40-1), 0.12% ortho-phenylphenol, sodium nitrite rust inhibitors, proprietary deodorizer, and 3.5% hydrocarbon propellant (personal communication, Calvin Goeder, Caltech Industries, 1988) and Lysol sprays contain 79.0% wt/wt denatured ethyl alcohol (SDA-40-1), 0.1% ortho-phenylphenol, rust inhibitors, N-alkyl-N-ethyl morpholinium ethylsulfate deodorizer, and carbon dioxide propellant (personal communication, Joe Rubino, MS, Lehn and Fink, 1988).

Concentration of ethyl alcohol appeared to be a critical factor. Other products with formulations similar to Citrace and Lysol sprays, but containing less ethyl alcohol (CoeSpray with 53.5% wt/wt ethyl alcohol and Pro-Cide ES with 52.8% wt/wt ethyl alcohol), failed to inactivate poliovirus in the presence of bioburden (Table

3). These findings led to special tests performed to investigate the antiviral activity of various concentrations of SDA-40-1 denatured ethyl alcohol used in Citrace and Lysol sprays in the presence of 10% whole blood. Results showed a dramatic increase in virucidal activity with alcohol concentrations equal to or greater than 70% wt/wt. Klein and Deforest[52] reported similar results with poliovirus I. Also, many other investigators have reported very rapid inactivation of both viruses and bacteria with ethyl alcohol in concentrations of 70%-95%.[32,57-65] However, clinicians and researchers can be misled about alcohol concentrations in commercial products if they do not understand the volume/volume and weight/weight designations.

Antimicrobial activity of high concentration ethyl alcohol can become unpredictable if storage conditions allow undetected volatilization, environmental use conditions cause extremely rapid evaporation, or interfering denaturing agents are used. Unfortunately, clinicians have no way to monitor these variables. Therefore, Citrace and Lysol sprays appeared better suited than ethyl alcohol alone for environmental surface disinfection in clinical settings because they are sealed in airtight cans to prevent volatilization during storage; they contain other ingredients in their formulations that delay evaporation during use; and their denaturing agents are standardized.

Currently, official agencies and others recommend use of iodophors,[42,66-71] chlorines,[5,42,66,69,72-75] and phenolics[75] for disinfection of environmental surfaces. Results from this investigation suggest further review of these recommendations. This work showed iodophors had very poor antimicrobial activity in the absence and

presence of bioburden, regardless of the test method or contact time used. Horse serum and paper towel material caused iodophors to lose almost all activity. Many others have reported problems with iodophor antimicrobial activity on inanimate surfaces.[52,63,76-81] With chlorines, the potential of bioburden to diminish antimicrobial activity has been mentioned by many investigators[52,63,78,82] and this problem was further demonstrated in this study. The failure of different phenolic formulations to inactivate poliovirus using both 3- and 10-minute contact times was also demonstrated, and this problem has been noted by others.[6,63,83-85]

In addition, this study confirmed previous reports citing inability of isopropyl alcohol to inactivate poliovirus,[52,86] problems with antimicrobial activity of dilute glutaraldehyde,[87] and inability of quaternary ammonium compounds to inactivate poliovirus[52,83,84] and the TB organism.[78,88]

Precleaning of surfaces before disinfectant use has been stressed.[41,70,71,73,89-94] In the past, detergents have been preferred for this process. The obvious intent is to decrease proteins and other debris that interfere chemically with the antimicrobial activity of disinfectants. Although theoretically this appears sound, use of cleaners with low antimicrobial activity before disinfectant application causes cleaning personnel to touch concentrated body fluids containing potential pathogens. It ignores the fact that the wiping action can spread material from smaller concentrated areas to larger areas, and onto the wipe material and the person performing the cleaning. Now that disinfectants have been identified that penetrate and kill microbes within heavy bioburden, it

seems prudent to apply these agents first to a lower organism loads before human contact. Disinfectants containing high ethyl alcohol and ortho-phenylphenol can be used first to preclean and the disinfect in the following regimen: wet surface well and allow 2-3-minute disinfectant contact time to lower viable microbial load within debris; wipe vigorously to clean surfaces; rewet surface and allow 2-3-minute contact with disinfectant after cleaning. Appropriate barriers should be worn by cleaning personnel when using the suggested method.

Four clinically relevant points were demonstrated in this study:

– *Disinfectants often have selective kill.* Although clinicians have been advised that tuberculocidal products can be depended on to kill other important pathogens,[95] this is not necessarily true. In this study, for example, 70% vol/vol isopropyl alcohol and the phenolic, Sporicidin Spray, produced profound kill of the TB test organism (*M bovis*), but failed to inactivate a resistant nonenveloped virus (poliovirus). This questions the assumption of broad-spectrum kill, based solely on any one organism.

– *Dilution generally decreases disinfectant activity.* This effect is seen in both chlorine and iodophor categories (Table 2). Although there are reports indicating dilution increases the antimicrobial activity of iodophors,[96] this study showed activity decreased as dilution was increased from 1:106 up to 1:213. The same was true of chlorines as dilution was increased from 1:5 up to 1:20.

– *Bioburden affects disinfectants adversely.* The deleterious effect of bioburden on antimicrobial activity was demonstrated repeatedly and with all disinfectants in this

study to varying extents. Citrace, Lysol sprays, and 70% vol/vol denatured ethyl alcohol were affected least.

– *Many commercial products have marginal activity.* This point was illustrated by the fact that 28 of the 39 products in this study failed one or more test organisms even in the absence of bioburden. Only three of the 11 products tested in the presence of bioburden inactivated all five test organisms, regardless of the test method used.

Reproducibility of the AOAC Use Dilution Method has been debated for many years. In this investigation, the 60-tube version of this test was performed 230 times on 39 different products representing eight different active ingredients. When results were arranged in order of disinfectant major ingredient (Table 2), it became apparent that the AOAC UDM was reproducible when disinfectants with definite high antimicrobial activity were tested. However, when disinfectants with marginal activity were tested, variability of results increased substantially. To see this pattern, it was necessary to test several representative products from each of eight major active ingredients.

Conclusions

Data from this investigation indicated that optimum disinfection of environmental surfaces was highly formulation dependent. Of the 39 products tested, only three inactivated all five test organisms, regardless of test conditions. The other products showed deficiencies that contraindicate their use, in the formulation tested, as environmental surface disinfectants in clinical dental settings.

Information about the manufacturers of the products mentioned in this article is available from the authors. Neither the authors nor the American Dental Association has any commercial interest in the products mentioned.

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Table 1 — Alphabetical listing by brand name of all disinfectants tested. The far right column indicates the concentration of active ingredients at the recommended use-dilution listed on the label.

BRAND NAME	RECOMMENDED DILUTION	USE CONCENTRATION OF ACTIVE INGREDIENT(S)
1. BASIC-G	1:256	0.05% quaternary ammonium compounds 0.01% tetrasodium EDTA 0.001% essential oils
2. BIOCIDE	1:213	82 ppm available iodine
3. BIOCIDE	1:106*	164 ppm available iodine
4. BORAXO	Use as packaged	9.13% tetrasodium EDTA 5.0% isopropyl alcohol 0.07% 5-chloro 2 (2,4-dichlorophenoxy) phenol
5. CITRACE	Use as packaged	66.6% ethyl alcohol 0.12% o-phenylphenol
6. CLOROX	1:5	1.05% sodium hypochlorite
7. CLOROX	1:10	0.52% sodium hypochlorite
8. CLOROX	1:20	0.26% sodium hypochlorite
9. CLOROX FRESH SCENT	1:5	1.05% sodium hypochlorite
10. CLOROX FRESH SCENT	1:10	0.52% sodium hypochlorite
11. CLOROX FRESH SCENT	1:20	0.26% sodium hypochlorite
12. COESPRAY	Use as packaged	53.46% ethyl alcohol 0.9% essential oils 0.176% o-phenylphenol 0.044% p-tert-amylphenol
13. DENTASEPTIC	1:32	0.28% o-phenylphenol 0.03% o-benzyl-p-chlorophenol
14. DISPATCH	Use as packaged	0.55% sodium hypochlorite
15. ETHYL ALCOHOL (non-denatured)	Use as packaged	70% v/v non-denatured ethyl alcohol
16. ETHYL ALCOHOL (denatured)	Use as packaged	70% v/v denatured ethyl alcohol
17. ETHYL:ISOPROPYL ALCOHOL MIX	Experimental	40% ethyl alcohol 40% isopropyl alcohol
18. ETHYL:ISOPROPYL ALCOHOL MIX	Experimental	45% ethyl alcohol 45% isopropyl alcohol
19. EXSPOR	1:1:4	1.43% organic acid 0.23% sodium chloride
20. HIBISTAT TOWELETTE	Use as packaged	70% isopropyl alcohol 0.5% chlorhexidine gluconate
21. HI-TOR	1:256	0.06% quaternary ammonium compounds
22. ISOPROPYL ALCOHOL	Use as packaged	70% v/v isopropyl alcohol
23. LYSOL LIQUID (Pine Scent)	1:103	0.16% soap 0.05% pine oil 0.04% o-benzyl-p-chlorophenol 0.015% isopropyl alcohol 0.007% tetrasodium EDTA
24. LYSOL SPRAYS (Regular Scent, Fresh Scent ... Light Scent, Professional Lysol)	Use as packaged	79% ethyl alcohol 0.1% o-phenylphenol
25. MATAR	1:256	0.04% o-benzyl-p-chlorophenol 0.02% o-phenylphenol 0.01% isopropyl alcohol 0.01% p-tert-amylphenol 0.007% tetrasodium EDTA
26. MULTICIDE	1:32	0.28% o-phenylphenol 0.03% o-benzyl-p-chlorophenol
27. OMNI II	1:32	0.28% o-phenylphenol 0.03% o-benzyl-p-chlorophenol
28. PERMACIDE 18	Use as packaged	18% ethyl alcohol 18% isopropyl alcohol
29. PERMACIDE 30	Use as packaged	30% ethyl alcohol 30% isopropyl alcohol
30. PRECISE	Use as packaged	0.37% o-phenylphenol
31. PRESEPT LIQUID	Use as packaged	70% v/v ethyl alcohol B 1.25% w/v chlorhexidine gluconate 0.1% w/v quaternary ammonium compounds
32. PRESEPT TABLET	1:250	1000 ppm available chlorine
33. PRESEPT WIPE	Use as packaged	70% denatured ethyl alcohol 1.25% chlorhexidine gluconate 0.1% quaternary ammonium compounds
34. PROCIDE ES	Use as packaged	52.79% ethyl alcohol 0.176% o-phenylphenol 0.044% p-tert-amylphenol
35. SPORICIDIN SPRAY	Use as packaged	18.0% ethyl alcohol 1.41% phenol 0.25% essential oils 0.24% sodium phenate
36. STERALL SPRAY	Use as packaged	1.50% triethylene-glycol 0.25%glutaraldehyde
37. VITAWIPES	Unknown	10.28% quaternary ammonium compounds
38. WESCODYNE	1:213	75 ppm available iodine
39. WESCODYNE	1:106*	150 ppm available iodine

*Tested at twice manufacturer's suggested strength.

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ENVIRONMENTAL SURFACE DISINFECTANTS

Disinfectants for environmental surfaces must kill *rapidly & thoroughly.* To identify products meeting these requirements, CRA is purchasing & testing disinfectants from all over the world. Following chart summarizes 24 alcohol-based products where only 4 passed all tests, 9 chlorine-based products where only 2 passed all tests, 1 cleaner which did not pass all tests, 5 iodophore-based products where none passed all tests, 10 phenolic-based products where none passed all tests, 1 quaternary ammonium chloride-based product which did not pass all tests, and 9 wipes where only 1 passed all tests.

MATERIALS & METHODS.

Tuberculosis bacteria & polio virus are used as test organisms because they are human pathogens with known resistance to kill by many disinfectants. Results are reported as $\log_{10}$ reductions of 10^7 test organisms exposed to disinfectants for 1, 3, & 10 minutes using 3 different tests performed in triplicate, without & with fresh human whole blood present. By convention, less than a 3 $\log_{10}$ reduction constitutes inadequate kill. Full methods are described in following publication: Christensen, R.P., et al., JADA '89; 119:493-505.

BLACKENED SQUARES ON CHART INDICATE INADEQUATE KILL

	DISINFECTANT NAME	COMPANY	COUNTRY	MAJOR ACTIVE INGREDIENT(S)	TEST TIMES	POLIO VIRUS I (Mahoney) SUSPENSION TEST WITHOUT BLOOD	POLIO VIRUS I (Mahoney) SUSPENSION TEST WITH BLOOD	TUBERCULOSIS ORGANISM (M. Bovis) SUSPENSION TEST WITHOUT BLOOD	TUBERCULOSIS ORGANISM (M. Bovis) SUSPENSION TEST WITH BLOOD	TUBERCULOSIS ORGANISM (M. Bovis) SURFACE TEST WITHOUT BLOOD	TUBERCULOSIS ORGANISM (M. Bovis) SURFACE TEST WITH BLOOD
	ALCOHOL-BASED										
	1. AMCIDE	Ashland	USA	62.8% Ethyl alcohol (w/w)	1 min	0.0	0.0	>5.0	4.8		
					3 min	1.0	0.1	>5.0	5.0	>5.8	4.3
					10 min	2.5	0.3	>5.0	4.8		
	2. ASEPTI-PHENE RTU	Huntingdon Laboratories	Canada	66% Ethyl alcohol (w/w)	1 min	0.8	0.1	4.8	4.1		
					3 min	1.7	0.4	5.0	3.8	5.3	3.4
					10 min	2.7	0.5	>4.8	4.2		
	3. BACTOL BLUE	Whiteley Chemical	Australia	70% Ethyl alcohol (w/w)	1 min	1.0	0.1	>4.1	4.8		
					3 min	3.1	0.2	>4.1	4.9	>4.5	3.8
					10 min	3.7	0.3	>4.1	5.3		
*	4. BIOSURF	Micrylium Laboratories	Canada	70.5% Ethyl alcohol (w/w)	1 min	4.7	3.0	4.8	5.2		
					3 min	>4.8	>3.7	5.0	5.4	4.6	3.6
					10 min	>5.0	>3.8	4.9	5.4		
	5. CITRACE	Caltech Industries	USA	68.4% Ethyl alcohol (w/w)	1 min	4.4	0.3	>4.7	4.4		
					3 min	4.4	1.0	>4.7	4.4	5.0	3.8
					10 min	3.9	1.5	>4.8	4.8		
	6. CITREX	Caltech Industries	USA	68.1% Ethyl alcohol (w/w)	1 min	4.1	0.9	>4.8	>4.8		
					3 min	>5.0	1.8	>4.8	5.2	>5.1	3.8
					10 min	>5.0	2.3	>4.8	5.2		
	7. COE SPRAY	GC America	USA	63% Ethyl alcohol (w/w)	1 min	0.6	0.0	>5.0	5.4		
					3 min	1.3	0.3	>5.0	5.7	5.3	4.0
					10 min	2.6	0.1	>5.0	>4.8		
	8. COE SPRAY "The Pump"	GC America	USA	64% Ethyl alcohol (w/w)	1 min	0.6	0.0	5.2	5.0		
					3 min	1.7	0.2	>5.1	4.9	>5.1	4.0
					10 min	2.5	0.3	5.4	4.8		
	9. CROSS PREP	Cregine	Australia	70% Ethyl alcohol (w/v)	1 min	0.5	0.0	5.4	4.3		
					3 min	1.1	0.0	5.4	4.8	>4.8	3.6
					10 min	1.9	0.0	>5.1	4.8		
	10. FD 320	Orochemie	Germany & Canada	42% 1-propanol (w/v) 10% Ethyl alcohol (w/v)	1 min	0.0	0.0	5.4	3.7		
					3 min	0.0	0.0	5.4	5.0	>5.6	4.7
					10 min	0.3	0.0	>5.1	5.0		
	11. FD 322	Orochemie	Germany & Canada	22% 1-propanol (w/v) 39% Ethyl alcohol (w/v)	1 min	0.0	0.0	5.7	4.8		
					3 min	0.2	0.0	>5.1	>4.8	>4.8	4.5
					10 min	0.7	0.0	5.4	>4.7		
	12. FORMAL SPRAY	Courtin	England	43.5% Ethyl alcohol (w/v) 19.6% 2-propanol (w/v) 4.0% 1-propanol (w/v)	1 min	0.2	0.0	>5.1	5.8		
					3 min	0.6	0.0	5.4	>5.5	>5.9	3.3
					10 min	1.0	0.0	>5.1	>5.5		
	13. GENERIC RUBBING ALCOHOL	Bergen Brunswig Drug	USA	70% Isopropyl alcohol (v/v)	1 min	0.0	0.0	5.3	4.9		
					3 min	0.0	0.0	>5.0	>4.8	5.2	3.7
					10 min	0.0	0.0	>5.0	>4.8		
	14. GERMX	Germiphene	Canada	76% Ethyl alcohol (v/v)	1 min	4.1	1.5	5.1	4.5		
					3 min	4.1	1.6	5.1	4.7	4.9	3.6
					10 min	4.0	2.6	4.8	4.6		
*	15. GLEN 20	Reckitt & Colman	Australia	68% Ethyl alcohol (w/w)	1 min	>5.3	3.7	5.1	5.2		
					3 min	5.5	>4.0	5.0	5.3	5.4	3.3
					10 min	>5.5	3.9	4.4	5.5		
	16. INCIDIN SPEZIAL-SPRAY	Henkel Hygiene	Germany	38.4% Ethyl alcohol (w/w)	1 min	0.0	0.0	2.3	2.6		
					3 min	0.0	0.0	5.3	4.4	1.7	1.3
					10 min	0.1	0.3	4.9	>5.5		
	17. LYSOL SPRAY (Germicidal)	Reckitt & Colman	Canada	64% Ethyl alcohol (w/w)	1 min	>5.0	1.6	5.6	5.9		
					5 min	>5.0	2.2	5.4	5.5	5.6	3.6
					10 min	>5.0	2.6	5.3	5.8		
*	18. LYSOL I.C. SPRAY	ProBrands Division of Reckitt & Colman	USA	79% Ethyl alcohol (w/w)	1 min	>5.2	>3.5	4.8	>4.8		
					3 min	>5.3	>3.4	4.6	4.9	5.0	3.9
					10 min	>5.2	>3.4	4.8	>4.8		
*	19. LYSOL II SPRAY	Reckitt & Colman	USA	79% Ethyl alcohol (w/w)	1 min	>5.7	3.4	4.8	>4.7		
					3 min	>5.8	3.1	>4.8	>4.7	4.4	3.8
					10 min	>5.8	3.8	>4.8	>4.7		
	20. MIKROZID	Schülke & Mayr	Germany & England	35% 1-propanol (w/w) 23.5% Ethyl alcohol (w/w)	1 min	0.0	0.0	5.2	5.5		
					3 min	0.0	0.0	5.1	>5.2	5.8	5.3
					10 min	1.0	0.1	5.0	5.8		
	21. SAGROMED HBV	Schülke & Mayr	Germany	35% 1-propanol (w/w) 23.5% Ethyl Alcohol (w/w)	1 min	0.0	0.0	5.3	5.4		
					3 min	0.1	0.0	>5.1	>5.2	>5.4	4.5
					10 min	0.9	0.0	>5.1	>5.2		
	22. SEP-T-BAN	McCormick Scientific	USA	68.9% Ethyl alcohol (w/w)	1 min	4.8	0.7	5.4	4.1		
					3 min	>4.7	1.3	5.2	4.3	5.3	4.2
					10 min	>4.8	1.8	5.9	4.4		
	23. SURFEX-E	Microtek Associes	Canada	70% Ethyl alcohol (w/w)	1 min	>4.4	0.3	>5.1	>5.2		
					3 min	>4.8	1.6	>5.1	5.4	5.2	2.8
					10 min	>4.8	1.8	>5.1	>5.2		
	24. VIRAHOL	Veridien	USA	70% Isopropyl alcohol (w/w)	1 min	0.0	0.0	5.3	>5.3		
					3 min	0.0	0.0	>5.3	>5.3	4.6	3.6
					10 min	0.0	0.0	6.0	>5.1		
	CHLORINE-BASED	NOTE: All Clorox tests included both the Household and Institutional brands. Since results from the 8 tests were similar, they were combined.									
*	1-2. CLOROX 1:2 (Household & Institutional)	Clorox Company	USA	2.63% Sodium hypochlorite	1 min	>5.2	>4.8	>5.8	>5.2		
					3 min	>5.2	>4.8	>5.1	>5.2	6.1	5.5
					10 min	>5.2	>4.8	>5.1	>5.2		
	3-4. CLOROX 1:5 (Household & Institutional)	Clorox Company	USA	1.05% Sodium hypochlorite	1 min	>5.2	>4.5	>5.1	1.6		
					3 min	>5.4	>4.5	>5.1	4.0	6.5	1.8
					10 min	>5.3	>4.5	>5.1	5.1		
	5-6. CLOROX 1:10 (Household & Institutional)	Clorox Company	USA	0.53% Sodium hypochlorite	1 min	>5.2	4.5	5.7	0.6		
					3 min	>5.2	>4.8	>5.2	0.8	6.3	1.3
					10 min	>5.0	>4.8	>5.2	1.7		
	7-8. CLOROX 1:100 (Household & Institutional)	Clorox Company	USA	0.05% Sodium hypochlorite	1 min	>4.8	1.3	>5.2	0.5		
					3 min	>4.4	1.3	>5.2	1.0	2.7	0.1
					10 min	>4.4	1.4	>5.2	1.6		
	9. EXSPOR 4:1:1	Alcide	USA	0.26% Sodium chlorite	1 min	>5.3	>4.4	>5.0	>5.2		
					3 min	>5.1	>4.3	>5.0	>5.2	3.0	0.1
					10 min	>5.2	>4.3	>5.0	>5.2		

* at left of product name indicates product passed all tests

OVER →

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BLACKENED SQUARES ON CHART INDICATE INADEQUATE KILL

DISINFECTANT NAME	COMPANY	COUNTRY	MAJOR ACTIVE INGREDIENT(S)	TEST TIMES	POLIO VIRUS I (Mahoney) SUSPENSION TEST WITHOUT BLOOD	POLIO VIRUS I (Mahoney) SUSPENSION TEST WITH BLOOD	TUBERCULOSIS ORGANISM (M. bovis) SUSPENSION TEST WITHOUT BLOOD	TUBERCULOSIS ORGANISM (M. bovis) SUSPENSION TEST WITH BLOOD	TUBERCULOSIS ORGANISM (M. bovis) SURFACE TEST WITHOUT BLOOD	TUBERCULOSIS ORGANISM (M. bovis) SURFACE TEST WITH BLOOD
CLEANER										
1. LYSOL I.C. FOAMING DISF/CLEANER not diluted	Reckitt & Colman	USA	0.1% Quaternary ammonium chloride	1 min	>4.8	0.3	0.1	0.0		
				3 min	>4.7	0.5	0.2	0.1	2.3	2.4
				10 min	>4.7	1.0	0.6	0.7		
IODOPHORE-BASED										
1. BI-ARREST (1:213)	Infection Control Technology	USA	75 ppm iodine	1 min	0.3	0.0	1.8	0.0		
				3 min	0.4	0.0	5.3	0.0	1.5	0.0
				10 min	1.9	0.1	4.8	0.0		
2. BIOCIDE (1:213)	Biotrol	USA	75 ppm iodine	1 min	0.3	0.0	1.8	0.0		
				3 min	0.4	0.0	5.3	0.0	1.5	0.0
				10 min	1.9	0.1	4.8	0.0		
3. IODOFIVE (1:213)	Cottrell, Ltd.	USA	75 ppm iodine	1 min	0.3	0.0	1.8	0.0		
				3 min	0.4	0.0	5.3	0.0	1.5	0.0
				10 min	1.9	0.1	4.8	0.0		
4. IODOPHOR DISINFECTANT (1:213)	Smart Practice	USA	75 ppm iodine	1 min	0.3	0.0	1.8	0.0		
				3 min	0.4	0.0	5.3	0.0	1.5	0.0
				10 min	1.9	0.1	4.8	0.0		
5. WESCODYNE (1:213)	Amsco	USA	75 ppm iodine	1 min	0.3	0.0	1.8	0.0		
				3 min	0.4	0.0	5.3	0.0	1.5	0.0
				10 min	1.9	0.1	4.8	0.0		
PHENOLIC-BASED										
1. ASEPTI-PHENE 128 (1:128)	Huntington	USA	6.00% o-phenylphenol 0.94% o-benzyl-p-chlorophenol 0.41% p-tert-amylphenol	1 min	0.0	0.3	1.2	0.3		
				3 min	0.0	0.3	4.8	2.6	3.7	0.5
				10 min	0.0	0.3	>4.9	>5.1	1.8	0.7
2. BI-ARREST II (1:256)	Infection Control Technology	USA	0.06% o-phenylphenol (K salt) 0.06% p-tertiary amylphenol (K salt)	1 min	0.0	0.2	1.8	0.4		
				3 min	0.0	0.3	5.0	3.0	4.0	0.9
				10 min	0.0	0.3	>4.9	>5.1	3.0	0.9
3. BIREX SE (1:256)	Biotrol Int'l.	USA	6.09% o-phenylphenol 6.03% p-tertiary amylphenol	1 min	4.1	0.3	0.2	0.0		
				3 min	>4.7	0.3	3.0	0.0	2.9	0.3
				10 min	>4.7	0.3	>5.1	0.0	1.1	0.2
4. LYSOL BULK (3:100)	Reckitt & Colman	USA	0.08% o-phenylphenol 0.08% o-benzyl-p-chlorophenol 0.66% other misc. active ingred.	1 min	0.0	0.2	1.4	0.0		
				3 min	0.0	0.3	5.4	0.2	4.1	1.1
				10 min	0.0	0.3	>5.1	0.9	4.6	0.9
5. LYSOL I.C.-DISINFECTANT (1:200)	ProBrands Division of Reckitt & Colman	USA	0.09% o-phenylphenol 0.09% o-benzyl-p-chlorophenol	1 min	0.0	0.9	2.1	0.1		
				3 min	0.0	0.7	>5.1	1.0	2.7	0.7
				10 min	0.0	0.7	>5.1	4.6	1.3	0.3
6. LYSOL I.C.-DISF/CLEANER (1:128)	ProBrands Division of Reckitt & Colman	USA	0.02% o-phenylphenol 0.06% o-benzyl-p-chlorophenol	1 min	0.0	0.7	0.8	0.2		
				3 min	0.0	0.5	4.5	2.2	3.3	1.1
				10 min	0.0	0.7	>5.2	5.1	0.7	0.6
7. LYSOL I.C.-PRESOAK not diluted	ProBrands Division of Reckitt & Colman	USA	0.55% o-phenylphenol 0.55% o-benzyl-p-chlorophenol	1 min	0.0	0.5	3.8	2.0		
				3 min	0.0	0.7	>5.2	4.4	>5.0	3.4
				10 min	0.3	0.7	>5.2	5.1	>5.1	4.9
8. PROCIDE SPRAY not diluted	Cottrell, Ltd.	USA	0.28% o-phenylphenol 0.03% o-benzyl-p-chlorophenol	1 min	0.0	0.3	0.9	0.0		
				3 min	0.0	0.4	5.4	0.4	3.8	0.9
				10 min	0.0	0.6	>6.2	3.0	2.8	0.7
9. SPORICIDIN not diluted	Sporicidin Int'l.	USA	1.86% Phenol 0.06% Sodium phenate	1 min	0.0	0.2	2.5	2.2		
				3 min	0.0	0.1	>5.2	4.2	>5.0	4.4
				10 min	0.1	0.2	>5.2	5.8	5.7	5.0
10. VITAL DEFENSE-D (1:32)	Block Drug	USA	0.28% o-phenylphenol 0.03% o-benzyl-p-chlorophenol	1 min	0.0	0.5	3.1	0.3		
				3 min	0.0	0.3	>6.2	3.2	4.4	1.8
				10 min	0.0	0.4	>6.2	>4.5	3.6	1.6
QUATERNARY AMMONIUM-BASED										
1. CAVICIDE	Karr	USA	0.25% Quaternary ammonium chloride 15.3% Isopropyl alcohol (w/w)	1 min			1.5			
				3 min	1.1	0.2	3.1	3.3	2.1	2.3
				10 min	>3.4	0.4	>5.5	>5.3		
WIPES										
1. ASEPTI-WIPE Pre-wetted	Ecolab Professional Products	USA	0.28% Quaternary ammonium chloride 8.0% Isopropyl alcohol (v/v)	1 min	0.0	0.2	0.0	0.0		
				3 min	0.1	0.0	0.0	0.1	0.0	0.1
				10 min	0.0	0.4	0.0	0.1		
2. METRIWIPES Pre-wetted	Metrex Research Corp.	USA	0.28% Quaternary ammonium chloride 8.0% Isopropyl alcohol (v/v)	1 min	0.0	0.2	0.0	0.0		
				3 min	0.1	0.0	0.0	0.1	0.0	0.1
				10 min	0.0	0.4	0.0	0.1		
3. SANI-CLOTH Pre-wetted	Nice-Pak PDI	USA	0.28% Quaternary ammonium chloride 8.0% Isopropyl alcohol (v/v)	1 min	0.0	0.2	0.0	0.0		
				3 min	0.1	0.0	0.0	0.1	0.0	0.1
				10 min	0.0	0.4	0.0	0.1		
4. DISCIDE ULTRA Pre-wetted	Palmero Healthcare	USA	0.34% Quaternary ammonium chloride 63.29% Isopropyl alcohol (w/w)	1 min	0.0	0.0	3.3	4.0		
				3 min	0.0	0.1	5.2	5.1	4.1	1.2
				10 min	0.0	0.1	>4.4	5.1		
* 5. INDI-WIPES (w/ Lysol Spray) † Not pre-wetted	Elrae Health Products	USA	3.17% Poly hydrochloride 7.11% Quaternary ammonium chloride	1 min	>3.8	>3.5	5.2	4.1		
				3 min	>3.5	>3.4	5.1	4.2	4.8	3.5
				10 min	3.7	>3.4	4.7	4.4		
† Indi-Wipes are a dry cloth impregnated with poly hydrochloride & quaternary ammonium chloride. The manufacturer's directions state, "Lightly dampen wipe with clean water or 70% alcohol." CRA tested the wipes by wetting with water or alcohol in separate tests. Results of wetting with 20 ml of water are below & results of a second group of tests wetting with 20 ml of Lysol Spray, whose active ingredient is 79% ethyl alcohol (w/w), are above. Commercially, there are no Indi-Wipes impregnated with Lysol Spray, but clinically they could be wetted thoroughly with Lysol Spray just before use & perform well.											
6. INDI-WIPES (w/ Water) † Not pre-wetted	Elrae Health Products	USA	3.17% Poly hydrochloride 7.11% Quaternary ammonium chloride	1 min	0.2	0.2	0.2	0.0		
				3 min	0.3	0.1	0.1	0.0	0.6	0.1
				10 min	0.3	0.2	0.2	0.0		
7. SPORICIDIN Pre-wetted	Sporicidin Int'l.	USA	1.86% Phenol 0.06% Sodium phenate	1 min	0.0	0.1	0.4	0.2		
				3 min	0.0	0.0	1.4	1.0	1.1	1.1
				10 min	0.7	0.0	4.9	3.9		
8. SUPER SANI-CLOTH Pre-wetted	Nice-Pak PDI	USA	0.5% Quaternary ammonium chloride 56% Isopropyl alcohol (w/w)	1 min	0.0	0.3	3.4	3.3		
				3 min	0.1	0.1	5.3	4.4	4.9	3.2
				10 min	0.0	0.1	5.1	4.4		
9. VIRAHOL Pre-wetted	Veridien Corp.	USA	79% Isopropyl alcohol (w/w)	1 min	0.0	0.1	4.7	4.5		
				3 min	0.0	0.0	4.7	4.7	1.5	0.1
				10 min	0.1	0.0	4.6	4.7		

* at left of product name indicates product passed *all* tests

CRA CONCLUSIONS: **Of 59 disinfectants tested only 7 passed all tests, & all of those were either ethyl alcohol or chlorine-based. Inability to inactivate a resistant virus was the most prevalent lack. Clinicians must be aware of kill potential of disinfectants, & avoid use of products that fail to kill resistant organisms from both bacterial & viral categories.**

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REFERENCE NO. 16

Clinical Research Associates Abstract
J. Dent. Res. 69:379 #2161 March 1990

Tuberculocidal Activity of Glutaraldehyde & Glutaraldehyde/Phenol Disinfectants. R. Robison, D. Robinson, B. Ploeger, & R. Christensen (Clin. Res. As. Provo, UT USA)

Recent studies have established the inability of glutaraldehyde (glut) disinfectants to kill *Mycobacterium tuberculosis* (TB) rapidly. This deficiency has been diminished by raising the glut concentration from 2% to 3.2% & by addition of phenols. The purpose of this study was to compare the tuberculocidal activity of several glut & glut/phenol disinfectants. Products tested were: Cidex7; Cidexplus; Metricide Plus 30; ProCide 30; Sporicidin diluted 1:2, 1:8, & 1:16; Sterall undilute (UD) & 1:4; & Wipe Out UD & 1:2. The EPA Tuberculocidal Activity Test Method (quantitative) was used & linear regressions were performed to establish the time needed for a 6 $\log_{10}$ reduction of *M. bovis* (ATCC #35743) for each disinfectant. All products were tested once a week for 6 weeks to determine tuberculocidal activity & amount of glut & phenol present. TB kill time ranges from initial use to 6 weeks, ordered most to least effective, were: Sporicidin 1:2 = < 1 min; Wipe Out UD = 3 to 4 min; MetriCide Plus 30 = 21 to 28 min; Cidexplus = 22 to 42 min; Procide 30 = 28 to 42 min; Cidex7 = 29 to 58 min; Sterall UD = 36 to 42 min; Wipe Out 1:2 = 35 to 63 min; Sporicidin 1:8 = 53 to 58 min; Sporicidin 1:16 = 192 to 254 min; & Sterall 1:4 = 340 to 399 min. *It was concluded that the tuberculocidal activity of combination glut/phenol preparations was superior to glut-*

only preparations & water dilution recommended by manufacturers affected both types of formulations adversely.

REFERENCE NO. 17

VOLUME 15, ISSUE 5-- MAY 1991
CRA NEWSLETTER

SUBJECT: DISINFECTANTS, INSTRUMENT IMMERSION

Today heat sterilization is required for critical instruments because it provides best possible margin of safety against cross contamination. However, some items will not tolerate heat, & submersion in liquid disinfectant is highest level of treatment possible. Clinicians have difficulty selecting disinfectants because they have no way to know if expected microbial kill is achieved. Although agencies such as EPA, FDA, & ADA have responsibility to monitor claims, none actually *test* products. Their registration & approval programs are based on data submitted by manufacturers. CRA questioned accuracy of glutaraldehyde & glutaraldehyde-phenolic claims because apparently similar products had very different claims for test organism kill times, optimum dilution, & reuse life. Therefore, it was decided to perform tests to determine relevance of current label & promotional information on glutaraldehyde-based instrument immersion disinfectants. Following report includes: (1) goals of CRA testing; (2) results of testing; & (3) CRA conclusions.

1. GOALS OF CRA TESTING.

Purpose of tests was to determine of differences existed among several glutaraldehyde-based disinfectants. 10 formulations sold under 12 brand names were tested at various dilutions (total of 18 solutions). Tests included EPA Tuberculocidal Activity Test

Method (using *Mycobacterium bovis* ATCC #35743) to evaluate tuberculosis (TB) claims & a conventional suspension test (using poliovirus I, Mahoney strain) to evaluate poliovirus (polio) claims. These 2 organisms were selected because of their known resistance to inactivation by glutaraldehyde. Viruses of current interest to dental clinicians, such as HIV & HBV, were not used as test organisms because of technical difficulties they present in tests & because they have been shown to be readily susceptible to inactivation by most common disinfectants. Organic stress was not used because it is not required by EPA to validate organism claims.

2. RESULTS OF TESTING.

Chart on pages 2-3 gives details of this evaluation. Below is summary of results.

(a) TUBERCULOSIS TEST RESULTS – 12 of the 15 solutions with a TB kill claim required longer contact times than listed on their label to produce EPA required reductions.

(b) POLIOVIRUS TEST RESULTS – 10 of the 15 solutions with a polio claim required longer contact times than listed on their label to produce EPA required reductions.

(c) TUBERCULOSIS PLUS POLIOVIRUS TEST RESULTS – 4 of the 18 solutions tested fulfilled EPA requirements for both TB & polio through the last day of their reuse life claim in less than 30 minutes (Sporicidin 1:2 & 1:4, Cidex plus, &

Metricide Plus 30), & 7 more fulfilled these criteria in less than 60 minutes (Cidex 7, Procide, Metricide 28, Wipe Out not diluted, Banicide & Sterall not diluted, & Sporicidin diluted 1:8).

(d) TUBERCULOSIS KILL DID NOT NECESSARILY INSURE INACTIVATION OF POLIOVIRUS & VICE VERSA – Clinicians have been lead to believe kill of the TB organism signals kill of all other pathogens. CRA has reported previously that this is not true (Christensen, et al., JADA 119:493 Oct. '89). It is critical to note that 5 solutions in this evaluation required time beyond TB kill time to inactivate polio (Sporicidin diluted 1:16, 1:8, 1:4; Wipe Out not diluted; & Wipe Out diluted 1:2).

3. *CRA CONCLUSIONS:*

(a) Disinfectants with similar active ingredients had equivalent performance despite diverse label claims.

(b) Clinicians need to increase contact times well beyond conventional 10 minutes to assure inactivation of resistant organisms.

(c) Increased contact times were always associated with increased dilution of active ingredients.

(d) All 18 solutions tested inactivated both test organisms to levels required by EPA, if clinician is willing to tolerate whatever contact time data indicate. CRA recommendations on specific brand names tested are listed on page 3.

TIME REQUIRED TO KILL TUBERCULOSIS & POLIOVIRUS TEST ORGANISMS
CRA test results vs. manufacturers' stated times

CRA contact times are means of multiple tests performed over the solution's reuse life. Add at least 15 minutes to all means for margin of safety to allow for clinical-use stress on solutions (i.e. organic load, dilution, aging).

BRAND NAME, COMPANY, & COST/OZ AT DILUTION TESTED	TIME	DISINFECTANT CATEGORY	DILUTION TESTED BY CRA	PERCENT ACTIVE INGREDIENTS AT DILUTION TESTED	MFR. STATED REUSE LIFE
1. SPORICIDIN 1:2 ‡ Ash/Dentsply Cost/oz @ this dilution = $1.66	TB Mfr rec: none CRA test: 1 m Polio Mfr rec: none CRA test: 1 m	Glutaraldehyde-Phenolic	Diluted 1 part H_20 by CRA §	1.00% glutaraldehyde 3.53% phenol 0.60% sodium phenate	No stated reuse life for this dilution
2. SPORICIDIN 1:4 ‡ Ash/Dentsply Cost/oz @ this dilution = 83¢	TB Mfr rec: none CRA test: 6 m Polio Mfr rec: none CRA test: 11 m	Glutaraldehyde-Phenolic	Diluted 3 parts H_20 by CRA §	0.50% glutaraldehyde 1.76% phenol 0.30% sodium phenate	No stated reuse life for this dilution
3. METRICIDE PLUS 30 Metrex Cost/oz @ this dilution = 16¢	TB Mfr rec: 20 m CRA test: 23 m Polio Mfr rec: 3 m CRA test: 6 m	Glutaraldehyde	Used as packaged per mfr. directions	3.20% glutaraldehyde	30 days
4. CIDEXPLUS J&J Medical Cost/oz @ this dilution = 22¢	TB Mfr rec: 20 m CRA test: 26 m ¶ Polio Mfr rec: 10 m CRA test: 6 m	Glutaraldehyde	Used as packaged per mfr. directions	3.20% glutaraldehyde	28 days
5. WIPE OUT Biotrol, Int'l Cost/oz @ this dilution = 18¢	TB Mfr rec: 15 m CRA test: 4 m Polio Mfr rec: 10 m CRA test: 35 m	Glutaraldehyde-Phenolic	Used as packaged per mfr. directions	0.30% glutaraldehyde 1.00% phenol as sodium phenate	45 days
6. CIDEX 7 J&J Medical Cost/oz @ this dilution = 22¢	TB Mfr rec: 90 m CRA test: 35 m ¶ Polio Mfr rec: 10 m CRA test: 6 m	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	28 days
7. METRICIDE 28 Metrex Cost/oz @ this dilution = 14¢	TB Mfr rec: 10 m CRA test: 36 m Polio Mfr rec: 10 m CRA test: 8 m	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	30 days
8. BANICIDE Pascal Cost/oz @ this dilution = 28¢	TB Mfr rec: 30 m CRA test: 44 m Polio Mfr rec: 10 m CRA test: 44 m ¶	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	42 days
9. STERALL Colgate/Hoyt Cost/oz @ this dilution = 22¢	TB Mfr rec: 30 m CRA test: 47 m Polio Mfr rec: 10 m CRA test: 43 m ¶	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	42 days
10. PROCIDE Cottrell, Ltd. Cost/oz @ this dilution = 20¢	TB Mfr rec: 10 m CRA test: 48 m Polio Mfr rec: 10 m CRA test: 7 m	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	28 days
11. SPORICIDIN 1:8 Ash/Dentsply Cost/oz @ this dilution = 41¢	TB Mfr rec: none CRA test: 50 m Polio Mfr rec: none CRA test: 53 m	Glutaraldehyde-Phenolic	Diluted 7 parts H_2O per mfr. directions	0.25% glutaraldehyde 0.88% phenol 0.15% sodium phenate	30 days
12. OMNICIDE Cottrell, Ltd. Cost/oz @ this dilution = 20¢	TB Mfr rec: 10 m CRA test: 64 m Polio Mfr rec: 10 m CRA test: 17 m	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	28 days

13. GLUTAREX 3M Cost/oz @ this dilution = 29¢	TB Mfr rec: 10 m CRA test: 70 m Polio Mfr rec: 10 m CRA test: 8 m	Glutaraldehyde	Used as packaged per mfr. directions	2.00% glutaraldehyde	28 days
14. COLDSPOR 1:20 Metrex Cost/oz @ this dilution = 16¢	TB Mfr rec: 10 m CRA test: 1.9 h Polio Mfr rec: 2 m CRA test: 1.4 h ¶	Glutaraldehyde-Phenolic	Diluted 19 parts H_2O per mfr. directions	0.50% glutaraldehyde 0.025% o-phenylphenol 0.005% p-tert-amylphenol	30 days
15. WIPE OUT 1:2 Biotrol, Int'l Cost/oz @ this dilution = 9¢	TB Mfr rec: 15 m CRA test: 72 m Polio Mfr rec: 10 m CRA test: 2.8 h	Glutaraldehyde-Phenolic	Diluted 1 part H_2O per mfr. directions	0.15% glutaraldehyde 0.50% phenol as sodium phenate	45 days
16. SPORICIDIN 1:16 Ash/Dentsply Cost/oz @ this dilution = 21¢	TB Mfr rec: 10 m CRA test: 3.4 h Polio Mfr rec: 10 m CRA test: 3.5 h	Glutaraldehyde-Phenolic	Diluted 15 parts H_2O per mfr. directions	0.13% glutaraldehyde 0.44% phenol 0.08% sodium phenate	30 days
17. BANICIDE 1:4 Pascal Cost/oz @ this dilution = 7¢	TB Mfr rec: 30 m CRA test: 5.7 h Polio Mfr rec: 10 m CRA test: 5.0 h	Glutaraldehyde	Diluted 3 parts H_2O per mfr. directions	0.50% glutaraldehyde	21 days
18. STERALL 1:4 Colgate/Hoyt Cost/oz @ this dilution = 6¢	TB Mfr rec: 30 m CRA test: 6.4 h Polio Mfr rec: 10 m CRA test: 5.9 h ¶	Glutaraldehyde	Diluted 3 parts H_2O per mfr. directions	0.50% glutaraldehyde	21 days

‡ = More concentrated than recommended § = CRA tested solutions more concentrated than company recommends for products that did not received 6 $\log_{10}$ TB & 3 $\log_{10}$ polio reduction within recommended contact times. ¶ = Cidexplus & Cidex 7 TB claims by manufacturer are for 25°C, & Sterall & Banicide polio claims by manufacturer are for 23°C. Higher temperatures would result in shorter contact times.

CRA OBSERVATIONS & RECOMMENDATIONS

CRA OBSERVATIONS –

(1) POTENTIAL REASONS FOR DIFFERENT CLAIMS OF APPARENTLY SIMILAR PRODUCTS – (a) Different efficacy test methods & parameters used; (b) Different laboratory technicians with different techniques; (c) Error within test methods; (d) Standard tests may not have been performed properly i.e. improper neutralization, improper organism challenge, no temperature control, etc.; & (e) Present registration system does not provide confirmation of disinfectant efficacy.

(2) NEED FOR DISINFECTANT MONITORS – Routine use of disinfectant monitors is necessary to tell clinician to discard solution when active ingredients have dropped to independable levels.

(3) NO IDEAL DISINFECTANT – All disinfectants tested exhibited one or more characteristics that can be undesirable such as fair to poor odor, corrosion of metal if soaked overnight, staining of skin &/or plastic instruments, special requirements for disposal, high cost, etc. Ideal disinfectant lacking in all negative characteristics does not exist. Clinicians must determine characteristics they can & cannot tolerate.

(4) EFFECTS OF WATER DILUTION – Undesirable clinical characteristics & costs are decreased by water dilution, but dilution also decreased antimicrobial activity which extended contact times significantly.

(5) BENEFITS & PROBLEMS WHEN GLUTARALDEHYDE COMBINED WITH PHENOLIC – Combination of glutaraldehyde with phenolics is attempt to take advantage of glutaraldehyde's

rapid virucidal activity & phenolic's rapid tuberculocidal activity. CRA tests showed concentrations of glutaraldehyde & phenolic are critical. Altering concentrations of either chemical can skew activity toward one organism at expense of other.

(6) REUSE LIFE CLAIMS VS. SOLUTION LEVELS – Under heavy use, solution levels can drop so low after about 2 weeks that longer reuse life claims become irrelevant.

CRA RECOMMENDATIONS –

Since all 18 disinfectants tested eventually destroyed both test organisms, clinical choices can be based on desired *speed of disinfection, amount of tolerable active ingredients, & cost.* CRA suggests following rationale for choosing product best suited to individual needs.

(1) *IF CLINICIAN NEEDS FASTEST POSSIBLE KILL OF RESISTANT ORGANISMS & COST, ODOR, & CORROSION ARE NOT CONSIDERATIONS* – Sporicidin 1:2 or 1:4 are products of choice.

(2) *IF CLINICIAN NEEDS FAST KILL OF RESISTANT ORGANISMS & TOLERANCE OF ORGANIC LOAD OVER TIME IS NEEDED* – Metricide Plus 30 or Cidexplus 3.2% glutaraldehydes are products of choice. These higher concentration glutaraldehydes are ideal for *pre*-disinfection of instruments to lower microbe counts before handling. CRA tests show higher glutaraldehyde concentration maintains activity longer during heavy use & in presence of heavy bioburden.

(3) *IF CLINICIAN WILL TOLERATE 45-60 MINUTE SOAK TIME INDUSTRY STANDARD IS 2% GLUTARALDEHYDE* – There are numerous 2% glutaraldehydes & glutaraldehyde-phenolics from which to choose (Banicide not diluted, Cidex 7,

Procide, Metricide 28, Sporicidin diluted 1:8, Sterall not diluted, & Wipe Out not diluted). For practices that can tolerate soak times from 1-3 hours, 2 additional 2% glutaraldehydes (Glutarex & Omnicide) & 2 additional glutaraldehyde-phenolics (ColdSpor diluted 1:20 & Wipe Out diluted 1:2) are possibilities.

(4) *WHEN SOAK TIMES CAN EXTEND 4-6 HOURS & LOWEST POSSIBLE CONCENTRATION OF ACTIVE INGREDIENTS IS DESIRED TO MINIMIZE HYPERSENSITIVITY* – Sporicidin diluted 1:16 & Banicide & Sterall both diluted 1:4 fill this need.

REFERENCE NO. 18

JADA, Vol. 129, July 1998

HOW WELL DOES THE CHEMICLAVE STERILIZE HANDPIECES?

ROBERT A. KOLSTAD, PH.D.

ABSTRACT

Using the Food and Drug Administration's protocol for testing health care sterilizers, the author investigated the ability of chemical vapor and steam to sterilize handpieces. Five internal sites of six high-speed handpiece models and four internal positions of one low-speed handpiece model were each inoculated with 10^6 *Bacillus stearothermophilus* spores. Half-cycle challenges were conducted with Chemiclave models EC 5500 and 8000 (Barnstead/Thermolyne) and with two autoclaves, Tuttnauer 2540M (large chamber) (Tuttnauer USA Co., Ltd.) and Statim Cassette (SciCan USA). Experiments with spores either openly exposed or partially enclosed prove that

Between-patient instrument processing – including cleaning, packaging, loading, device operation, monitoring and storage – is a subject of continuous interest to heath care practitioners.[1-6] So, too, is sterilization, which dental offices experience as procedure, not end effect. The practicing dental community does not inspect instruments cycled through steam, heated air or chemical vapor for surviving microorganisms.

The marketing of sterilization devices is regulated by the Food and Drug Administration, or FDA. In 1976, that

agency began developing an official definition of sterilization, requiring manufacturers intending to market health care sterilization devices to prove efficacy claims.[7] The FDA published its requirements in 1993.[8] In that document, the agency prescribed the boundaries of the sterilization process, incorporating two basic principles.

– **Overkill.** After use, each instrument is assumed to be contaminated with microbes higher in both number and resistance than those actually encountered. Accordingly, efficacy studies are to be initiated with one million (10^6) microbes of the kind most difficult to destroy by the lethal agent in question. For example, bacterial spores could be used in a study of the effectiveness of steam or heated air.

– **Sterility assurance level.** The sterility assurance level, or SAL, is the probability – by consensus considered low enough for patient safety – that each processed item bears a single surviving microbe. For reused health care instruments, the SAL is one in one million (10^{-2}).

Sterilization claims are expressed as the stress needed to achieve the prescribed viability reduction – for example, the length of time an instrument is exposed to a particular temperature. In practice, microbes on suitable carriers – such as stainless steel washers – are exposed to steam, heated air or another lethal agent. The sterilization claim is the time during which the population is reduced to 10 percent multiplied by 12 (the time during which viability would fall from 10^6 to 10^{-6}). In turn, that rate of extrapolation must be confirmed with a test employing instruments that bear 10^6 microbes. The exposure time

during which total kill is achieved must not exceed half the sterilization claim.

In 1995, the FDA embarked on an initiative – still in draft form[9] – complementary to the 1993 ruling. It stated that handpiece manufacturers must validate sterilization claims through microbial testing closely related to confirmatory challenges mandated in the 1993 publication. This study was conducted to determine the ability of chemical vapor and steam to sterilize rotary instruments according to the FDA handpiece testing protocol.

MATERIALS AND METHODS

Microbial materials. I obtained *Bacillus stearothermophilus* American Type Culture Collection, or ATCC, 7953 spores in aqueous suspension from SPS Corp., $2x10^8$/milliliter $D_{121\ C}$=1.9 minutes tested on a paper carrier.

The incubation medium I used was Trypticase Soy Broth (Becton-Dickinson), or TS broth. Sporulation medium consisted of Nutrient Broth (Difco Laboratories) plus 0.005 grams per liter of manganese sulfate solidified with 2 percent agar 20 mL per petri plate.

Equipment. Six each of six high-speed handpiece models were used: Midwest Tradition L and Midwest Quiet Air (Dentsply Midwest), Lares 557 and Lares 757 (Lares Research), KaVo 642B (KaVo America Corp.) and Star 430SWL (Den-Tal-Ez Inc.). Twelve low-speed handpieces Star 62398 (Det-Tal-Ez Inc.)were employed. All handpieces were drawn from manufacturers' new inventory.

The chemical vapor sterilizers I used were Chemiclave EC 5500 (representing the nonvacuum line) and Chemiclave 8000 (prevacuum/post vacuum line). The lethal agent Vaposteril (Barnstead/Thermolyne), which I used in both chemical vapor sterilizers, includes the active ingredients ethanol 72.38 percent and formaldehyde 0.23 percent. For all Chemiclave models, the manufacturer's stated maximum temperature is 132 C. With the aid of an Omega model COCO001 thermocouple (Omega Engineering Inc.), I validated that temperature. Pressurized steam equipment included the large-chambered Tuttnauer 2540M (Tuttnauer USA Co., Ltd.), whose operational temperature – 121 C and 134 C – were adjusted per thermocouple, and the small-chamber Statim Cassette autoclave (SciCan USA), whose operational temperature – 135 C to 137 C – could be thermocouple-measured but not adjusted.

For biological challenges, the Chemiclaves were preheated with a dummy cycle, to minimize microbial destruction during warmup. Timing began when chamber pressure reached 20 pounds per square inch. I also ran a dummy cycle with the Statim autoclave. Timing began at 135 C. In contrast, I did not prewarm the Tuttnauer autoclave. The Tuttnauer's timing begins when the chamber door is locked; thus, microbes are exposed to the lowest total lethal exposure when the device starts out "cold".

Research design. The research design was developed in accordance with the FDA's Guidance Document on Dental Handpieces.[9] The FDA's designated test organism

for steam is *B. Stearothermophilus* ATCC 7953. For Chemiclave validation, the FDA makes no specific recommendation. However, based on a study that compared three bacterial species' resistance to heated Vaposteril,[10] the Chemiclave manufacturer selected *B. Stearothermophilus* as the test microorganism. As mentioned earlier, I used *B. Stearothermophilus* ATCC 7953 for Chemiclave challenges.

The FDA handpiece document calls for inoculating unsealed internal locations with 10^6 microbes each, after which inoculated instruments are exposed to one-half the nominal sterilization cycle. The minimum sample size is three handpieces per challenge run, with three runs per handpiece type.

Handpieces brought into the study from manufacturers' stock were clean; instruments in testing emerged from incubation either sterile or heavily contaminated with vegetative microbial growth. To ensure that all handpieces started out clean, I prepared them for experimental trials by exposing them to high-quality (97 percent dry) steam in a laboratory-grade autoclave, rinsed them in tap water, scrubbed them for three minutes in a Ney model 250 ultrasonic bath (Neytech Inc.) containing liquid detergent, rinsed them in distilled water and dried them.

High-speed handpiece trials. After initial sterilization and cleaning, handpieces were readied as though for clinical use, according to manufacturers' directions.

– The chuck, the drive air line and the exterior surfaces of the Midwest handpieces were sprayed with Midwest Plus Handpiece Cleaner. These handpieces were brush-scrubbed in running tap water, rinsed, run without water

until excess cleaner was expelled, then dried. They were lubricated with three drops of Midwest Plus Handpiece Lubricant that were placed in the drive air line. They then were run without water until excess oil was expelled.

– Lares handpieces were brush-scrubbed in running water, rinsed, dried, and cleaned and lubricated from the base with Lares Handpiece Conditioner. They were run 60 seconds with air and water. Lares handpieces were cleaned and lubricated both before and after heat exposure.

– KaVo handpieces were brush-scrubbed in running water, dried, and cleaned and lubricated from the base with KaVo Spray.

– Star handpieces were brush-scrubbed with isopropyl alcohol, then dried.

Contamination involved deposition of 0.01 mL spore suspension per site. A microsyringe was used for inoculation. In one experimental series (conducted with large lumens), turbines were removed and inoculated on one or more blades. In addition, drive air lines and exit air lines were inoculated. After the parts were dried for one hour at 37 C, turbines were returned to their housing. In an alternative test series (conducted with small lumens), chip air lines and cooling water lines were inoculated, then dried one hour at 37 C.

Contaminated handpieces were either subjected to no prechallenge handling or processed according to manufacturers' directions. Contaminated handpieces sealed individually in paper pouches obtained from the Chemiclave manufacturer were exposed to heat, three per

cycle. Three runs were conducted per handpiece type and protocol step. Half-cycles were 10 minutes (Chemiclaves), five minutes (Statim), 17.5 minutes (Tuttnauer low temperature) and 12.5 minutes (Tuttnauer high temperature).

After exposure, handpieces were immersed in 80 mL TS broth in 100-mL graduated cylinders and incubated at 56 C until growth appeared or seven days had elapsed (step H in Figure 1). Growth signifies recovery of one or more viable spores. To ensure that growth was *B. Stearothermophilus*, two cultural characteristics were controlled. During its multiplication in TS broth, *B. Stearothermophilus* ATCC 7953 displays uniform turbidity. As growth ceases, a white pellet gathers at the container base; the medium above is clear. Second, a sample of microbial suspension was spread on TS agar and incubated for 40 hours at 56 C. After this, vegetative rods, rods containing endospores and free spores could be observed through a phase-contrast microscope.

Low-speed handpiece trials. After their initial sterilization and cleaning, handpieces were inoculated at four positions, each with 0.01 mL spore suspension. Sites included the nose cone chuck; input drive air port, forward chuck rotation; input drive air port, reverse chuck rotation; and the exit air port. Inoculated motors were dried at 37 C for one hour.

To determine whether packaging affects sterilization, I exposed contaminated handpieces, some open and some sealed in pouches, to heat, three instruments per challenge. Three runs were conducted for each of the two packaging modes. The Statim autoclave and Chemiclave

model EC 5500 were preheated with dummy cycles. Timing began as in the high-speed handpiece trials. The Statim half-cycle for unpackaged loads is 105 seconds and for packaged items, five minutes. The Chemiclave half-cycle is 10 minutes.

After being exposed, handpieces were immersed in 32 mL TS broth in glass tubes 25 millimeters x 150 mm and incubated at 56 C until growth appeared or seven days had elapsed. Control for *B. stearothermiphilus* was conducted as in the high-speed handpiece trials.

Accidental contamination control. To demonstrate that microbes surviving heat exposure were not chance contaminants but were included in the deliberate inoculum, modified low-speed challenges were conducted. As a negative control, 12 handpieces were processed as outlined above. Six instruments were transferred to TS broth and incubated at 56 C for seven days. No growth ensued. The other six handpieces were packaged, then subjected to half-cycle exposure in Chemiclave model EC 5500, removed from packaging and incubated in TS broth for seven days. No growth ensued. In the positive control, 12 handpieces were sterilized and cleaned. The handpieces were sterilized again, inoculated, packaged and exposed to half-cycle Chemiclave vapor, then incubated. Eight incubation tubes showed growth, all typical of *B. Stearothermophilus*.

Washers. To independently investigate Vaposteril lethality and penetration, I conducted challenges with spores on exposed surfaces and deep within tubes. Stainless steel washers 13 mm in diameter were inoculated with 0.01 mL spore suspension. Samples were dried at 37

C for one hour, then exposed to heat in a Chemiclave model EC 5500. Timing commenced at 20 psi.

In one set of trials, five washers resting on a wire-mesh screen were subjected to one, two, three, four, and five minutes' exposure. In another challenge series with the same timings, five glass tubes 15 x 125 mm, each holding one washer at the bottom, were placed in the chamber with the open end at an angle 30 degrees above horizontal. Loaded tubes were not capped. Exposed samples were incubated in TS broth at 56 C for seven days.

RESULTS

High-speed handpieces: Chemiclave tests. Initial half-cycle exposure involving contamination of all five internal sites resulted in spore outgrowth. Succeeding trials, conducted to more closely identify any sites that might offer protection, differentiated large lumens (air drive, turbine, air exit) and small lumens (air chip line and cooling water line). Both Chemiclave models successfully destroyed the biological challenge in the air-propulsion tract. Model EC 5500 was less effective than model 8000.

In retrospect, lubrication played no role in the outcome; no surviving spores were recovered from lubricated surfaces.

High-speed handpieces: autoclave tests. Initial trials at all five loci with the Statim device resulted in no microbial growth. Complete testing – large vs. small lumens – with the Statim and Tuttnauer autoclaves, the latter at both 121 C and 134 C, resulted in no surviving growth.

Low-speed handpieces. Four openly accessible locations were inoculated, and I made no attempt to discover which could be protective. In each of the two Chemiclave challenges, seven instruments emerged bearing viable spores; packaging appeared to play no role. In contrast, the Statim autoclave fully destroyed the bioburden, both in the faster unpackaged mode and the packaged mode.

Washers. The high-speed handpiece test results prove that chemical vapor can kill *B. Stearothermophilus* spores, and they suggest that narrow passageways thorough which the lethal agent must traverse could restrict contact with the biological target. The washer test separated lethality and access. Metal was chosen as a carrier, because handpiece lines are so constructed and because the material basis of a carrier greatly influences resistance of borne spores.[11] Clearly, Vaposteril is an effective killing agent. At one-minute exposure all samples emerged sterile.

In the enclosure trials, open tubes were positioned close to horizontal to best allow lethal agent penetration. Even that measure failed to overcome access limitation. It should be noted that autoclave exposure destroys spores in open tubes. The tube experiments demonstrate a discontinuous relationship between time in device (a better term than "exposure time") and lethal effect; moreover, time in device necessary to achieve even a partial effect was very long, relative to open exposure efficiency.

DISCUSSION

This study demonstrates that chemical vapor is an excellent sporicidal agent whose lethal activity is throttled by

small high-speed handpiece lines and motor internal locations. Challenges conducted with contaminated washers – either openly exposed to the lethal agent or sequestered at the closed end of glass tubes – strongly support the thesis that when chemical vapor fails, that failure is attributable to weak penetration, not killing power. So does experience with sterilizer operation. Considerably more time (and heat) is required to convert water to steam than to convert liquid Vaposteril to vapor. The higher specific heat of steam likely accounts for its greater penetration.

Well before the FDA began registering health care sterilizers, the Chemiclave was approved for marketing by the Environmental Protection Agency, which requires that equipment discharging potentially hazardous chemicals (in this case, formaldehyde) pass a stringent biological challenge involving spores of two bacterial genera borne on both hard and soft surfaces.[12] The hard carrier – small porcelain cylinders – is meant to represent instrument surfaces. Somehow the critical distinction between surfaces and instruments became lost; thus was born the myth that Chemiclaves sterilize all dental instruments. Downstream, that unproven assertion was to be recited as fact in risk management courses, journal and trade articles, infection control books, dental school lectures, expert conferences and other venues. The federal services, military and civilian, did decertify Chemiclaves, but the reason was formaldehyde toxicity. Handpiece manufacturers provide strong backing for the use of Chemiclaves by recommending it in package inserts. In fact, those manufacturers have operational integrity in mind, not pathogens: users are warned against using heat

beyond 135 C. Finally, endorsement for Chemiclaves comes from sterilization monitoring services, which unwittingly lead their subscribers to believe that spore-strip or other biological-indicator destruction signals load sterility.

The fundamental concept of health care sterilization shifted from surrogates to instruments when the FDA issued requirements for sterilizer marketing. That agency prohibits manufacturers from making claims for instruments without direct proof. Chemiclave models 7000 and 8000 are FDA-registered for handpiece sterilization, but those models are no longer sold. Chemiclaves now on the market are registered with the FDA, but without handpiece claims. The FDA does not specify instruments to be included in certification. Moreover, the agency prohibits manufacturers from informing the practicing dental community that their devices are FDA-registered. Obviously, that subtlety is lost on the practicing dental community, which cannot be expected to decipher arcane interplay between government and industry.

CONCLUSION

This study should have an impact on dental sterilization policy. For offices in which the sole sterilizer is a Chemiclave, I advise ordering and autoclave for high-speed handpieces and low-speed attachments. Motors can be protected with plastic sleeves and autoclaved periodically. Reusable three-way syringe tips are best replaced with disposable tips. Until the autoclave arrives, a pressure cooker operated with distilled water should suffice for sterilizing handpieces and motor attachments.

For its part, the Chemiclave manufacturer might wish to enhance chemical vapor penetration, either by modifying the Vaposteril formula or by redesigning the equipment and its operation.

This study was conducted with testing less stringent than that required for FDA sterilizer certification. I endorse no equipment. In selecting an autoclave, the practitioner should verify that the device has been registered with the FDA and bears a claim for whatever instrument the practitioner recognizes as most difficult to sterilize. Generally, this means high-speed handpieces.

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No. 98-1536

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In The

SUPREME COURT OF THE UNITED STATES

October Term 1998

Randon Bragdon, D.M.D.,

Petitioner,

v.

Sidney Abbott, et al.,

Respondents.

ON PETITION FOR A WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE FIRST CIRCUIT

PETITIONER'S SUPPLEMENTAL BRIEF

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PETITIONER'S SUPPLEMENTAL BRIEF

Petitioner Randon Bragdon, D.M.D., has petitioned for review of, inter alia, what level of risk is legally significant under the direct threat provisions of the Americans with Disabilities Act ("ADA"), and whether health care workers are required by the ADA to confront greater risks than patients or other citizens. On page 21, note 5, and page 28, of his petition, Dr. Bragdon cites *Onishea v. Hopper*, 126 F.3d 1323 (11th Cir. 1997), *vacated for en banc review*, 133 F.3d 1377 (11th Cir. 1998). On April 7, 1999, the Eleventh Circuit issued its en banc decision in *Onishea v. Hopper*, ___ F.3d ___ , 1999 WL 190763 (11th Cir.). The en banc *Onishea* decision is significant for Dr. Bragdon's petition for several reasons.

I. *ONISHEA* FURTHER ILLUSTRATES A CONFLICT AMONG THE CIRCUITS ON WHAT LEVEL OF RISK IS LEGALLY SIGNIFICANT UNDER THE ADA.

The ADA defines a direct threat as a significant risk to the health or safety of others that cannot be eliminated by reasonable modifications of policies, practices, or procedures, or by the provision of auxiliary aids or services. 42 U.S.C. § 12182(b)(3); 28 C.F.R. § 36.208(c). *Onishea* further illustrates a conflict among the circuits on what constitutes a direct threat under this significant risk standard.[1] As *Onishea* explains, *see Onishea*, 1999 WL 190763, *6-*7, the Fourth, Fifth, Sixth, and now the Eleventh Circuits, follow a cautious rule on how low the odds may be, and how much evidence it takes to prove a significant risk, when the adverse event is contraction of a fatal disease or death generally. On the other hand, the First Circuit and arguably the Ninth Circuit take a

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[1] Like other courts, *Onishea* recognizes the direct threat standard is the same under both the Rehabilitation Act of 1973 and the ADA. *Onishea*, 1999 WL 190763, *6 n.14; *see also* Pet. App. 20a-21a (tracing origin of direct threat standard to *Arline*, 480 U.S. 273 (1987)).

substantially less cautious approach. *Compare Montalvo v. Radcliffe*, 167 F.3d 873 (4th Cir. 1999); *Doe v. University of Md. Med. Sys. Corp.*, 50 F.3d 1261 (4th Cir. 1994); *Leckelt v. Board of Comm'rs of Hosp. Dist. No. 1*, 909 F.2d 820 (5th Cir. 1990); *Bradley v. University of Tex. M.D. Anderson Ctr.*, 3 F.3d 922 (5th Cir. 1993), *cert. denied*, 114 S. Ct. 1071 (1994); *Estate of Mauro v. Borgess Med. Ctr.*, 137 F.3d 398 (6th Cir. 1998), *cert. denied*, 119 S. Ct. 51 (1998) *with Abbott v. Bragdon*, 163 F.3d 87 (1st Cir. 1998) *and Chaulk v. United States Dist. Ct.*, 840 F.2d 701 (9th Cir. 1988). District Courts in the Third and Eighth Circuits have also followed the more cautious approach. *See Scoles v. Mercy Health Corp. of S.E. Pa.*, 887 F. Supp. 765 (E.D. Pa. 1994); *Doe v. Washington Univ.*, 780 F. Supp. 628 (E.D. Mo. 1991).

II. DR. BRAGDON CLEARLY RAISES A GENUINE ISSUE FOR TRIAL UNDER *ONISHEA'S* DIRECT THREAT STANDARD.

Onishea is one more example of a court applying a direct threat standard under which Dr. Bragdon would clearly deserve a trial, if that standard were applied to him. The Eleventh Circuit reasoned and held as follows:

> . . . [S]ignificance by itself connotes more than size: "significant" means "deserving to be considered," "important," "weighty," or "notable." And it is the potential gravity of the harm that imbues certain odds of an event with significance. . . .
>
> Thus, when the adverse event is the contraction of a fatal disease, the risk of transmission can be significant even if the probability of transmission is low: death itself makes the risk "significant."
>
> . . . .
>
> . . . . Accepting proof of a theoretically possible method of transmission as sufficient

> recognizes—unlike the First Circuit's approach—that saying a risk of an event is small does not mean that it will not happen. And each time the event occurs, it is real people—not cold ciphers—that suffer the consequences. Saying that only one in 42,000 persons will die if exposed to a risk, *see Mauro*, 137 F.3d at 407, does not make that one's death insignificant.
>
> We thus hold that when transmitting a disease inevitably entails death, the evidence supports a finding of "significant risk" if it shows both (1) that a certain event can occur and (2) that according to reliable medical opinion the event can transmit the disease. This is not an "any risk" standard: the asserted medical danger of transfer must be rooted in sound medical opinion and not be speculative or fanciful. But this is not a "somebody has to die first" standard, either; evidence of actual transmission of the fatal disease in the relevant context is not necessary to a finding of significant risk.

Onishea, 1999 WL 190763, *6-*8.

Dr. Bragdon's evidence clearly satisfies this standard. For example, CDC's seven reported cases of possible occupational HIV transmission to dental workers by themselves establish both that the event can occur, namely, percutaneous occupational exposure of dental workers to HIV-infected blood, and that the event can transmit the disease. *See* Petition pp. 14-16 and Pet. App. 224a, 549a-550a. The affidavit testimony of Dr. Sanford Kuvin, M.S., M.D., D.T.M. & H., also by itself establishes the event can occur and the event can transmit the disease. *See* Pet. App. 606a (1 in 66,667 risk per intra-oral injection); *see also* Pet. App. 573a-575a, 602a-610a.

From a qualitative standpoint, in 1993, Centers for Disease Control and Prevention ("CDC") characterized the HIV risk from dentists to patients as "small," Pet. App.

530a, and experts on both sides in this case agree the risk to Dr. Bragdon is at least as great and perhaps "much greater" than the risk to patients, Pet. App. 87a, 191a, 246a, 570a, 581a. Under the qualitative standard for significant risk set by the Eleventh Circuit, such a small-to-much-greater-than-small risk to dentists would be significant.

Similarly, in 1988, and again in 1997, professional articles written for dentists characterized their occupational HIV risk as "low." *See* Carol Chenoweth, et al., *Postexposure Chemophrophylaxis for Occupational Exposure to HIV in the Dental Office*, 128 J. Am. Dental Assoc. 1135, 1136 (Aug. 1997); Klein, et al., *Low Occupational Risk of [HIV] Infection Among Dental Professionals*, 318 New England J. of Med. 86 (Jan. 1988). This professionally recognized level of occupational HIV risk to dentists also meets the Eleventh Circuit's qualitative standard for significance when the adverse event is contracting HIV.

And again, in 1987, CDC warned dentists that universal precautions should be the "minimum precautions" for all dental procedures in which the potential for bleeding exists, thereby recognizing an HIV risk "deserving to be considered" and "notable" despite taking universal precautions. *See* Pet. App. 459a. Under the Eleventh Circuit's standard, this qualitatively "notable" and "deserving to be considered" HIV risk to dentists is also significant, or at least generates an issue for trial in light of Ms. Abbott's private, expert affidavits that the risk is "infinitesimal."[2]

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[2] To Dr. Bragdon's knowledge, no CDC official has ever said the risk to dental workers is "infinitesimal." This includes the affidavits from Dr. Wayne Marianos in this case, which, in any event, the Court agreed may not be used against Dr. Bragdon, *see* Pet. App. 22a. Only Ms. Abbott's private, **non**-public health official experts, Dr. Molinari and Dr. Craven, have offered affidavit opinions that the risk is

It is also noteworthy that, today, CDC takes the position that the risk of HIV transmission to a dental worker who sustains even a "superficial scratch" from an HIV-contaminated instrument is so great he or she should immediately begin four weeks of painful chemo-prophylaxis to reduce his risk of contracting HIV by some unknown factor. *See* Pet. App. 192a-193a, 195a-197a, 201a-204a (four week "basic regimen," as defined at Pet. App. 214a, is recommended for an "EC Code 2" (which includes superficial scratches) from an "HIV SC 1" (asymptomatic HIV-infected patient)), 208a-209. In light of this present day CDC concern for superficial scratches, the view there is no genuine issue for trial that Dr. Bragdon reasonably believed there was a significant risk is incomprehensible.

III. CONTRASTS BETWEEN THE PROCESS AND RESULTS IN *ONISHEA* AND *BRAGDON* ARE A STRIKING REMINDER OF WHY THIS COURT SHOULD EXERCISE ITS SUPERVISORY POWERS IN THIS CASE.

A. Prison Inmates Are Entitled to the Benefit of a Trial and Extensive Findings of Fact, But Dental Workers Are Not.

The HIV-infected prisoners who brought suit in *Onishea* went to trial. At trial, they had the opportunity to present live, thorough, testimony from their experts and other witnesses, and to cross-examine the defendants' experts and other witnesses in front of the fact finder. The

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"infinitesimal." To Dr. Bragdon's knowledge, no professional article or similar source of information published for dentists, before or after 1994, ever characterized the HIV risk to dental workers as "infinitesimal." Published sources assess the risk as "small," "low," or "very small." *See* Pet. App. 569a (reporting in 1993 that an American Dental Association study suggested "that dentists are at small risk of occupationally acquired HIV infection"); Chenoweth, *supra*, 128 J. Am. Dental Assoc. at 1136 (characterizing the risk as "low").

prison inmates had an opportunity to present their case, though not even as fully as the Eleventh Circuit majority thought proper, holding as it did that some of the plaintiffs' evidence was wrongly excluded at trial. Based on the live presentation of evidence at trial, the district court made extensive findings of fact contained in a "476-page opinion [sic]," *Onishea*, 1999 WL 190763 at *4, *8 n.16.

In contrast, Dr. Bragdon has had no trial and there have been no findings of fact. Later, on appeal, the First Circuit somehow expected Dr. Bragdon's attorney to explain in oral argument in less than a few minutes what it would take a medical expert several hours to explain under the best of circumstances. Thereupon summary judgment was affirmed against Dr. Bragdon in a conclusory, three-page appellate opinion. Though technically incorrect, impartial observers uneducated in the law might conclude from the disparity in process between *Onishea* and *Bragdon* that prison inmates simply have greater procedural rights than dental workers, and that prison life is worthy of extremely careful factual consideration, whereas the concerns of dental workers are not.

B. The Lives of Prison Inmates in Alabama are More Valuable Than the Lives of Dental Workers in Maine.

The Eleventh Circuit clearly takes a more cautious approach than the First Circuit on the issue of HIV transmission. For example, by upholding the district court's findings, the Eleventh Circuit, in effect, affirms the view that the risk of HIV transmission from an HIV-infected prison inmate, to non-infected inmates in "clerical classes, . . . floral design classes, . . . nutrition classes, concerts and talent shows," would be significant and could not be rendered insignificant by reasonable accommodations. *Onishea*, 1999 WL 190763, *1. In other words, in Alabama, because of the HIV-risk to uninfected prisoners from HIV-infected students in a floral design class, those with HIV may be excluded under federal law. In contrast,

in Maine, federal law requires dental workers in non-emergency situations to take mortal risks equivalent to jumping out of a plane with a parachute. *Compare* Pet. App. 606a (risk of accidental contraction of HIV when giving intra-oral injection is approximately 1 in 66,667 *with* Pet. App. 596a (risk of fatal accident while skydiving in 1992 is approximately 1 in 100,000).

The terrible but unavoidable conclusion, frankly put, is that federal law in the Eleventh Circuit puts a higher value on the lives of prison inmates than the First Circuit puts on dental workers. Small or low HIV risks to prison inmates are legally significant, but similarly small or even greater risks to dental workers are not.

CONCLUSION

The Court should grant certiorari to bring sense to a senseless conflict among the circuits on what level of risk is legally significant and, unfortunately, whose lives appear to count for more in a direct threat analysis. The Court can accomplish this goal by reiterating "Who is at risk?" is not a proper factor in the direct threat analysis and by remanding for a trial under an appropriate qualitative or quantitative standard of significance similar to the one articulated by the Eleventh Circuit.

At the same time, Dr. Bragdon's support for the Eleventh Circuit's approach does not mean he agrees with all the particular results reached in *Onishea*. Even under a properly cautious approach to direct threat, it is not clear to Dr. Bragdon that HIV-infected prison inmates really pose a direct threat to other inmates in all of the programs listed in *Onishea*, e.g., horticultural classes, clerical classes, floral design classes, college classes, and so forth. To the extent there is no direct threat in some of those instances, the problem is not the standard for significant risk set by the Eleventh Circuit. The problem, instead, appears to be a failure to genuinely apply the standard on an individualized basis both by program and by prison inmate involved. Of course, it may also be that such individualized

assessments are not judicially feasible, or administratively feasible in a prison environment.

The important point is that like the Eleventh Circuit, Dr. Bragdon is not, and has never argued for a "no risk" standard whereby normal classroom situations or other casual contacts with HIV-infected persons might be deemed significant risks. Otherwise stated, while Dr. Bragdon would have no qualms hugging HIV-infected persons in his office, it is a different matter manipulating needles and other sharp instruments in the back of their mouths and putting his face in an aerosol mist of their bloody saliva. For the latter, Dr. Bragdon desired to exercise additional precautions, beyond universal precautions, as 1987 CDC Guidelines said he should. Pet. App. 459a.

Wherefore, Dr. Bragdon respectfully requests that the Court grant his petition for a writ of certiorari and give him an opportunity at trial to show he reasonably assessed the risk as significant based on medical and objective evidence available in September 1994.

Respectfully Submitted,

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No. 98-1536

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In The

SUPREME COURT OF THE UNITED STATES

October Term 1998

Randon Bragdon, D.M.D.,

Petitioner,

v.

Sidney Abbott, et al.,

Respondents.

ON PETITION FOR A WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE FIRST CIRCUIT

PETITIONER'S REPLY BRIEF

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PETITIONER'S REPLY BRIEF

Petitioner Randon Bragdon, D.M.D., replies to respondents Sidney Abbott and Maine Human Rights Commission ("Respondents") as follows:

First, Respondents do not attempt to deny the undeniable, namely, that the 1987 CDC Guidelines literally say the opposite of what the Appeals Court says they say. Instead, as Respondents have done all along, they simply assert there is a wealth of evidence to support them, without citing one, single, solitary, concrete example of any affidavit, article, public health official guideline, or anything else in the entire universe, that was available to dentists in September 1994, which would convince a reasonable dentist there was no significant risk using universal precautions.

Second, Respondents do not address the extremely problematic conflict among the circuits on what level of risk is legally significant, or whether the lives of health care workers are less valuable than patients, prison inmates, or other citizens. These are important issues of law worthy of the Court's attention.

Third, contrary to Respondents, the lower courts have not reviewed theses issues on three occasions. The District Court applied an incorrect legal standard. The Appeals Court incorrectly relied on the 1993 CDC Guidelines and the American Dental Association policy statement. Now the Appeals Court incorrectly relies on the 1987 CDC Guidelines and unilateral assertions made for the first time by the American Dental Association in its amicus brief on remand in 1998. It is noteworthy that Robert J. Gherardi, B.S., D.M.D., FAGD, FACD, FADI, Vice-Chairman of the American Dental Association's Council on Dental Therapeutics in 1991, and a member of the Association's Reference Committee on Scientific Matters in 1992, has filed a *amicus* brief in support of Dr. Bragdon. *See* Br. of Amicus Curiae Congressman Charles Norwood, D.D.S., et al., at 16a.

Fourth, Respondents make a series of specious procedural arguments, all raised for the first time. For example, they now object that the article at 318a was not part of the record before the District Court. However, it was. *See* Response by Randon Bragdon to motion for Summary Judgment, p. 3, n.4 (citing, among others, Tokars, et al., to support Dr. Kuvin's use of a 0.3% infectivity rate in his calculations) (docketed Oct. 6, 1995).

Then, according to the most brazen double-standard of all, Respondents are content to have the Appeals Court rely on assertions made by the American Dental Association for the first time in its amicus brief on remand in 1998, but object to the letters at 414a and 416a, written in 1990 by the American Dental Association Council-on-Dental Therapeutics, advising the American Association of Forensic Dentists that the Council could find no scientific support that gloves and other infection control barrier techniques effectively reduce the transmission of microorganisms.

The Court must also keep in mind that at the District Court level, neither party introduced evidence for summary judgment on the theory there would be a legal distinction made between pre-September 1994 and post-September 1994 sources of information. In the original appeal, the Appeals Court reached this issue and stated a correct legal standard that post-September 1994 information should not be used against Dr. Bragdon, but the case was not remanded for the introduction of evidence to comport with the new legal standard. Thereafter, again, one of the difficult consequences of this Court's remand to the First Circuit, rather than to the District Court (and later the First Circuit's denial of Dr. Bragdon's motion to permit experts to sort out the pre- and post-1994 evidence), is that experts have never been given a chance in this case to focus on what was available before September 1994. Nevertheless, the indisputable fact is that the article at 418a and the article quoted on page 15 of the petition are important examples of information written for and available to dentists in 1994, which would have led them

reasonably to believe universal precautions do not eliminate significant risks. Though Respondents did not object to these articles on remand, they now object to the Court's consideration of them during the petition process. *See also* Br. of Amicus Curiae Clinical Research Associates at 2, 18 ("Failure of infection control products that support the universal precautions concept virtually negates the concept" and "CRA's data indicate that there *is* a significant risk, not overcome by universal precautions, every time HIV positive patients receive treatment where bleeding tissue and use of sharp instruments is involved").

Fifth, Respondents raise again a specious argument already implicitly rejected by the District Court and the Appeals Court, namely, that in filing and arguing for his own motion for summary judgment in the District Court, Dr. Bragdon somehow waived the argument there could be genuine issues for trial. As any first year civil procedure student should understand, the argument that when evidence is viewed most favorably for the plaintiff, no reasonable person could find for her, necessarily entails the view that when the evidence is viewed most favorably for the defendant, reasonable persons could find for him. *See* 10A Charles A. Wright, et al., *Federal Practice and Procedure* § 2720 at 16-17 (1983) ("the fact that both parties simultaneously are arguing there is no genuine issue of fact does not establish that a trial is unnecessary thereby empowering the court to enter judgment as it sees fit"); *id.* § 2725 at 104-108 (a party moving for summary judgment "is not entitled to a judgment merely because the facts he offers appears more plausible . . . or because it appears that the adversary is unlikely to prevail at trial. This is true even though both parties move for summary judgment.").

Attached is the full argument of counsel quoted out of context by Respondents. When pressed by the Court, it is clear that the thrust of counsel's argument was precisely that "when viewed in the light most favorable to the plaintiff, no reasonable person could conclude that there was not a

significant risk." *See* appendix pages 1,2,7 of this reply. Under rudimentary principles of civil procedure, this does not empower the District Court to grant summary judgment if there are genuine issues of fact. And of course, to this day, Dr. Bragdon believes he should have been granted summary judgment based on the undisputed expert testimony that the risk to him is as great or greater than to his patients, and the agreement among the Fourth, Fifth, Sixth, and Eleventh Circuits, and district courts in the Third and Eighth Circuits, that the risk to patients is legally significant despite universal precautions.

CONCLUSION

There are important issues of law at stake concerning the value of human life and whether patients and other citizens are more valuable than health care workers. The Appeals Court's opinion on remand is more patently infirm than the one already vacated by the Court. Four members of the Court already agree Dr. Bragdon clearly deserves a trial. Three were uncertain. Dr. Bragdon respectfully asks the Court to decide.

Respectfully Submitted,

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ABBOTT v. BRAGDON

Transcript of Proceedings
Hearing on Motions
U.S. District Court, District of Maine
October 26, 1995

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MR. SINGER: Your Honor, I'm Brent Singer for the defendant, Dr. Bragdon. I'd like to say just a few things and, of course, then I'll be happy to entertain questions from the court.

In ruling on the plaintiff's motion for summary judgment, the court must of course view the evidence in the light most favorable to Dr. Bragdon and see if a jury would be permitted from that to find that there is significant risk. And indeed, that's the issue in this case, is whether or not there's a significant risk to Dr. Bragdon when he treats a patient who is HIV-positive.

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And the CDC, despite what they have said, has not taken the position on the issue of significant risk. And its findings and its writings strongly imply that there is a significant risk, although the CDC has not expressly taken that position.

For example, the CDC has an article in the September '95, Journal of the American Dental Association, "JADA" for short. It would have been a perfectly appropriate time for the CDC in that article to inform dentists like Dr. Bragdon that they don't have to worry because the risk is insignificant, because it's infinitesimal, because it's negligible, etc. But that article does not say anything like that, and that's an article on

the risk to dentists from percutaneous injuries while performing basic procedures on health care workers.

Dr. Marianos is not the CDC. He is equivalent to someone who works for the EPA, even a high level EPA official, coming to the court and telling the court what the EPA meant to say in its official documents, but what it didn't actually say in its official documents.

Now we, of course, believe that, when the evidence is viewed in the light most favorable to the plaintiff, a jury would be compelled to find that there is a significant risk. That's one of the bases of our motions for summary judgment.

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For example, according to the CDC, there are 46 CDC-documented health care workers – it was 42, but more recent publication shows 46 – 46 CDC-documented health care workers who have HIV or have already died because of accidental exposure on the job to HIV-infected patients, plus an additional 97 other health care workers, including six dentists, for whom no risk factors have been identified by the CDC.

THE COURT: The plaintiff says that health care workers are judged on a different standard. We shouldn't be too concerned about that from the standpoint of HIV patients. You want to approach that argument or discuss it?

MR. SINGER: I see no legal basis for the conclusion that under the statute significant risk means one thing for Dr. Bragdon and another thing for patients. As stated in the brief, I think the plaintiffs invite the court to engage in a kind of calculus here where you begin to deem some lives more valuable than others. They argue, of course, that, because Dr. Bragdon is a dentist, he has somehow assumed this risk. We

do not think that's the case, but even those people in our society who undertake dangerous jobs and professions, it's not the role of the government then to force them to continue doing so.

THE COURT: Well, there hasn't been any finding by

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anyone that this is dangerous, is there?

MR. SINGER: The performance of invasive procedures –

THE COURT: In a dentist's office? Have you got any evidence to support that conclusion?

MR. SINGER: If I could quote, for example, to look at the CDC's 1993 guidelines on infection control in the dental office, not what the plaintiff say they say, but the guidelines themselves. It's an interesting document. It begins by describing the risk of transmission to dentists from infection of HIV in the office. It describes some elaborate and, according to the document itself, sometimes ineffective barrier precautions. It says – it concludes only that this, quote, should reduce the risk, which is obvious since, if you put on a pair of gloves, they're going to help somewhat. But that additional information is needed for an accurate assessment and that such information may lead to safer practices.

Now our position is that that implies that there must be some significant risk to the dentists because, otherwise, what are they doing in these articles? Why don't –

THE COURT: Let me ask you this. I mean, Mr. Webbert seems pretty clear about the fact that there doesn't seem to be any question at all that the CDC

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determination is that, in fact in a quotation form Mr. Marianos, that no infection control procedures beyond the use of universal precautions are necessary when providing routine dental care to persons with HIV and AIDS. And the affidavits and information contained in the material that's been presented to me by the plaintiffs would seem to lead to a pretty clear consensus that, as far as the CDC is concerned, there's no problem.

Are you suggesting that that's an – that's not necessarily an accurate recapitulation of what the evidence really is?

MR. SINGER: I'm suggesting that you need to read the CDC sources carefully and statements abound like "this can be done safely in the dental office." Well, as we point out, one can parachute safely with a parachute, one can perform invasive dental procedures in a dental office on persons with HIV. The issue is not whether or not it can be done safely; the issue is whether or not there's a significant risk in doing it. And that's what the CDC has not spoken on, and that is a glaring, absolutely glaring omission in their official published work.

Indeed, Dr. Marianos, unlike the other experts for the plaintiff, does not in his affidavits claim the risk is negligible or infinitesimal. As a spokesperson from the CDC, he goes about as far as saying that no additional precautions

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are necessary, which doesn't imply that they're not advisable. He goes as far as to say that it can be done safely, which doesn't say that it is safe.

Indeed, the plaintiff's – I'm sorry, the attorney for the United States has argued before the court today on several occasions that the CDC says that it is safe, but the CDC does not say it is safe.

THE COURT: So, who is going to make that determination in this case? Are you suggesting then that there may very well be a factual issue as to whether or not the procedure is safe and whether or not the CDC says it's safe, and that that determination should be made by a fact finder in this case?

MR. SINGER: Well, I think, fortunately, the court has some guidance at the summary judgment stage from their own experts. Numerous courts for good reasons have determined that the risk from health care provider to patient is significant, including the risk from dental practitioner to patient. And we know that HIV has been transmitted from the dentist to the patient, and then their own expert in print and under oath states that the risk is much greater from patient to dentist.

THE COURT: Talk about the eyes of the beholder.

Now you want me to make a determination, if I understand you correctly, as a matter of law on summary

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judgment that just the opposite is the case. So, here I have the plaintiff coming before me and saying, first of all, you have your instructions, Judge Brody, from the United States Supreme Court, and you know who that is, and the United States Supreme Court says, You look up the very clear policy of the CDC and, when you look it up, because I've attached to it my memorandum, you'll see that it says no problem. And

because it says no problem, summary judgment has to issue for the plaintiff.

Then you come along and say you're not sure. In fact, you're saying the CDC may be saying just the opposite if you analyze their statements carefully and find the right ones, and you want me to say – I assume you want me to also follow the Unites State Supreme Court, right? And that means the first stop is the CDC, right?

MR. SINGER: (Nodding in the affirmative.)

THE COURT: And you're saying that, at that stop, the information isn't quite so clear, but it's certainly clear enough for the defendant to get summary judgment. Isn't that where I am?

MR. SINGER: Our position, your Honor, -- it's actually shocking when you read the CDC material itself the extent to which it supports Dr. Bragdon's position rather than the plaintiff's position. And when you say – when you look at all these sources that say no problem, we were

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under – they give the – the plaintiffs give the impression that, somehow out there, there is a multitude of official CDC publications or guidelines saying this. But if you look at the record, it is the defendant by and large who is inviting the court and attaching original sources and so forth for the court's attention.

Now, the significance of the risk, as I say, follows we think as a matter of law from their CDC's – from cases that have deferred to the CDC.

THE COURT: Can I interrupt you for a minute, because at some point we're going to have to make a determination here, both in these summary judgment motions and ultimately, if necessary, at trial. Is it you position, is it the defendant's position that this issue can be resolved as a matter of law by summary judgment one way or the other? That there is no factual issue to be resolved?

MR. SINGER: Absolutely. That's the basis of our motion for summary judgment, which Mr. McCarthy is prepared to speak with more directly to those points.

THE COURT: So we don't need a jury determination as to this issue because you're suggesting there isn't any factual issue. It can be decided as a matter of law.

MR. SINGER: Well, it can be decided as a matter of law because no – on this record, when viewed in the light most favorable to the plaintiff, no reasonable person could

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conclude that there was not a significant risk. It's obvious there's a significant risk.

THE COURT: Well, it's just as obvious to the plaintiff that there isn't. I mean, you heard his argument. Pretty direct argument, wasn't it?

MR. SINGER: Our evidence is based upon court cases that have found significant risk and expert testimony from their experts that –

THE COURT: Court cases that have found significant risk in this kind of situation?

MR. SINGER: Absolutely. They have found that the risk of transmission of HIV in an invasive dental procedure from dentist to patient is so significant that the dentist can lawfully be prohibited from performing a single additional invasive procedure. Their experts say, including the expert from CDC, acknowledges that the risk is at least as great, some of their experts say greater, to Dr. Bragdon. So, we have a difficult time understanding how there could be a genuine issue of fact regarding the significance of risk to Dr. Bragdon.

THE COURT: Thank you.